This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

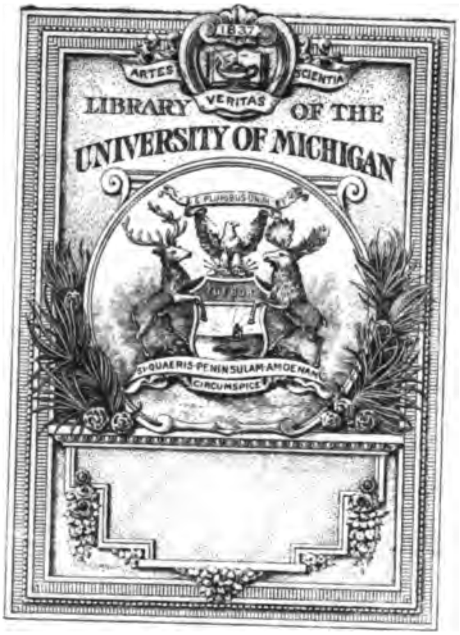
+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`



1837
ARTES VERITAS SCIENTIA
LIBRARY OF THE
UNIVERSITY OF MICHIGAN
E PLURIBUS UNUM
TUEBOR
SI QUAERIS PENINSULAM AMOENAM
CIRCUMSPICE

6.08
H

The Practice of Medicine

BY

H. V. HALBERT, M. D.

SENIOR PROFESSOR OF CLINICAL MEDICINE IN HAHNEMANN MEDICAL
COLLEGE CHICAGO; ATTENDING PHYSICIAN TO COOK
COUNTY HOSPITAL, CHICAGO.

PUBLISHERS:
P. H. MALLEN CO., CHICAGO
1905

COPYRIGHTED, 1905
BY
H. V. HALBERT

PREFACE.

My object in writing this book has been to present to our profession a concise work on the theory and practice of medicine which shall be, so far as possible, up to date. To fully accomplish this aim is a task which I had not truly estimated before the work was undertaken, else I fear in the rush of a busy practice I might have renounced the venture. To write a book requires not alone uninterrupted opportunity, but it must be done with despatch lest the subject matter be out of date before the ink of the publisher is dry. If my wish were to be gratified, I would delay the publication yet another year in order to present the subject more completely. It seems best, however, to hold to the original idea to offer this more as a concise text-book on practice for the student and busy practician, leaving the larger works already before us to cover the field of reference and more extensive detail. There are two classes of books which are demanded by the medical profession today. One is the comprehensive treatise which shall cover the minute investigations of experts in the line of pathology and the more intricate details of medical science; the other is a work which gives the essentials of etiology and pathology, with special emphasis on diagnosis and treatment. This has been the spirit which has actuated my writing this condensed practice. My only hope is that my object may be understood and that the work will be of some service to those whom I have tried to reach.

My next purpose has been to present a book which shall, so far as justifiable, meet the present demand for a broad view in the line of treatment. It is impossible for me to write and not express the liberal idea which is in harmony with my practice. I am thoroughly convinced that the homeopathic principal of practice is a correct one, for it refers to a symptomatology, gained by the proving of remedies, which is expressive of a real pathologic condition. More than that, when guided by the symptomatology in the selection of our remedies we are treating the patient as well as the disease. The success

139897

of the practician who heeds this law of therapeutics and at the same time accepts and applies any proven therapeutic discovery has been demonstrated by actual practice. We are open to censure if we do not avail ourselves of every therapeutic agent which has endured the test of thorough trial. To my mind no physician is properly equipped for practice without this comprehensive knowledge of medicine. Wherein a specific line of treatment has been demonstrated as valuable I believe it is the duty of an unbiased practician to use it unless he knows of a better method. In my teaching I have aimed to keep abreast of the times, and I trust this is apparent in my book.

The arrangement of subjects and the plan of composition which have been followed in this work are for the most part in line with standard text-books already known to the profession. In some instances it has seemed best to change the classification of diseases in accordance with recent views as to their cause. For this reason there is no chapter on the diseases of the muscles. Some of these have been included under the so-called "probable infectious diseases" and others have been mentioned under nervous diseases.

The publication of this volume would not have been possible had it not been for the liberty I have taken to quote and acquire from the writings of others. In this respect I am deeply indebted to Dr. W. C. Goodno. His Practice of Medicine and other writings, as well as his acquaintance, have been an inspiration and a help from the beginning. The excellent Practice by Anders has furnished me an object lesson in style and context. This I have followed freely and have often quoted from it. Osler, Tyson, Hare, Leube, Bartlett, Cowperthwaite, Lawrence, Clifford Mitchell's Diseases of the Urinary Organs and other authors have been freely consulted. For the chapter on nervous diseases I have relied mainly upon the standard works of Church and Peterson, Dana and Bartlett, while reference has been freely made to many other works on this subject.

I am also deeply indebted to Dr. E. M. Bruce and Dr. Clifford Mitchell for the review of the chapter on diseases of the urinary system and for their valuable advice on these subjects. To Dr. Vance Rawson I owe much for his careful revising and correction of manuscript and proof. Without his valuable aid in this respect the book might never have been finished. I wish also to speak of the valuable help rendered to me, in proof-reading and otherwise, by Dr. G. M. McBean. To my colleagues who have given me friendly suggestions and to my students who have encouraged me to undertake this work, I owe more than words can tell. My only hope is that the book will be accepted for what it is intended, and that my critics will be as considerate as possible.

H. V. Halbert.

70 State St., Chicago. October 10, 1905.

CONTENTS.

INFECTIOUS DISEASES.

- Typhoid fever 1
- Paratyphoid fever 39
- Mountain fever 40
- Typhus fever 40
- Relapsing fever 46
- Malta fever 49
- Malarial fever 50
- Dysentery 66
  - Catarrhal Dysentery 66
  - Bacillary Dysentery 67
  - Amebic Dysentery 69
  - Chronic Dysentery 70
- Cholera 75
- Yellow Fever 81
- Dengue 87
- The Plague (Bubonic) 91
- Cerebro-spinal Meningitis 93
- Erysipelas 103
- Septicemia 109
- Pyemia 112
- Rheumatic Fever 115
- Gonorrheal Arthritis 127
- Diphtheria 128
- Variola 138
- Vaccination Disease 146
- Varicella 147
- Scarlet Fever 149
- Measles 158
- Rubella 162
- Whooping Cough 164
- Mumps 169
- Influenza 171
- Lobar Pneumonia 178
- Tuberculosis 196
  - Tuberculosis of the Lymph-Glands 202
  - Acute Tuberculosis 204
  - Acute Pneumonic Phthisis 210
  - Chronic Pulmonary Tuberculosis 212
  - Fibroid Phthisis 223
  - Tuberculosis of the Alimentary Canal 225
  - Tuberculosis of Various Tissues 226
  - Tuberculosis of the Serous Membranes 226
  - Tuberculous Pleuritis 227
  - Tuberculosis of the Pericardium 227
  - Tuberculosis of the Peritoneum 228
  - Tuberculosis of the Liver 229
  - Tuberculosis of the Genito-Urinary System 230
  - Tuberculosis of the Testes, Prostate Gland and Seminal Vesicles 231
  - Tuberculosis of the Mammary Glands 231

- Tuberculosis of the Brain 231
- Tuberculosis of the Heart and Bloodvessels 232
- Treatment of Tuberculosis 234

Syphilis 240
- Visceral Syphilis 245
- Syphilis of the Brain and Cord 245
- Syphilis of the Liver 247
- Syphilis of the Alimentary Canal 248
- Syphilis of the Lungs 249
- Syphilis of the Circulatory System 250
- Syphilis of the Kidneys 250
- Syphilitic Orchitis 250
- Syphilis of the Joints 250

Leprosy 254
Glanders 256
Actinomycosis 258
Anthrax 260
Hydrophobia 262
Tetanus 266

Probable Infectious Diseases.

Muscular Rheumatism 270
Acute Multiple Myositis 273
Progressive Ossifying Myositis 274
Chronic Articular Rheumatism 274
Weil's Disease 276
Febricula 277
Milk Sickness 278
Miliary Disease 279
Foot and Mouth Disease 280
Glandular Fever 281

CONSTITUTIONAL DISEASES.

Diabetes Mellitus 283
Diabetes Insipidus 297
Arthritis Deformans 300
Gout 306
Lithemia 315
Rachitis 318
Osteomalacia 324
Scorbutus 326
Infantile Scorbutus 329
Purpura 331
Hemorrhagic Diseases of the New-born 335
Hemophilia 336
Obesity 338

DISEASES OF THE BLOOD AND DUCTLESS GLANDS.

Anemia 342
- Symptomatic or Secondary Anemia 342
- Primary or Essential Anemia 345
- Chlorosis 345
- Progressive Pernicious Anemia 350
- Leukemia 355
- Pseudo-leukemia 360
- Status Lymphaticus 364
- Splenic Anemia 365
- Anemia Infantum Pseudo-leukemia 367
- Chloroma 368

Diseases of the Ductless Glands.

Diseases of the Suprarenal Capsule 368
  Addison's Disease 368
Diseases of the Thyroid Gland 372
  Thyroiditis 372
  Goiter 372
  Myxedema 376
  Myxedema associated with congenital or acquired cretinism 378
  Neoplasm of the Thyroid 380

DISEASES OF THE RESPIRATORY SYSTEM.

Diseases of the Nose.

Acute Rhinitis 381
Chronic Rhinitis 383
Hay Fever 385
Epistaxis 387

Diseases of the Larynx.

Acute Laryngitis 388
Chronic Laryngitis 391
Spasmodic 393
Tuberculous 395
Syphilitic 397
Edematous 398
New Growths in the Larynx 399

Diseases of the Bronchi.

Bronchitis 400
Acute Bronchitis 400
Chronic Bronchitis 404
Bronchiectasis 409
Bronchial Stenosis 413
Asthma 414
Fibrinous Bronchitis 420

Diseases of the Lungs.

Circulatory Disturbances in the Lungs 422
Active Hyperemia 422
Passive Hyperemia 423
Hypostatic Hyperemia 423
Pulmonary Edema 424
Pneumorrhagia 429
Pulmonary Embolism 429
Chronic Interstitial Pneumonia 430
Broncho-pneumonia 432
Emphysema 435
  Compensating Emphysema 435
  Hypertrophic Emphysema 436
  Senile Emphysema 439
Gangrene of the Lungs 440
Abscess of the Lungs 441
Pneumokoniosis 443
Pulmonary Carcinoma 444
Hydatids of the Lungs 446

Diseases of the Pleurae.

Pleurisy 446
Acute Plastic Pleurisy 447
Sero-fibrinous Pleurisy 449
Empyema 460

Chronic Pleurisy 462
Pneumothorax 464
Hydrothorax 467
Neoplasms of the Pleura 468

Diseases of the Mediastinum.

Tumors of the Mediastinum 470
Inflammation affecting the Mediastinum 471
Mediastinal Hemorrhage 472
Diseases of the Thymus Gland 472

DISEASES OF THE HEART AND BLOODVESSELS.

Diseases of the Pericardium.

Pericarditis 473
Acute Plastic or Fibrinous Pericarditis 474
Sero-fibrinous Pericarditis 476
Purulent Pericarditis 483
Adhesive or Chronic Pericarditis 484
Hydropericardium 486
Hemopericardium 487
Pneumopericardium 487

Diseases of the Heart.

Endocarditis 488
    Simple Acute Endocarditis 488
    Ulcerative Endocarditis 493
    Chronic Endocarditis 496
Mitral Insufficiency 498
Mitral Stenosis 502
Aortic Insufficiency 505
Aortic Stenosis 509
Tricuspid Insufficiency 512
Tricuspid Stenosis 515
Pulmonary Valve Insufficiency 517
Pulmonary Valve Stenosis 517
Combined Valvular Lesions 518
Cardiac Hypertrophy 533
Cardiac Dilatation 540
Myocarditis 544
Acute Myocarditis 544
Chronic Myocarditis 546
The Fatty Heart 552
Atrophy of the Heart 555
Cardiac Aneurysm 555
Rupture of the Heart 556
Cardiac Neuroses 557
    Palpitation 557
    Tachycardia 560
    Bradycardia 564
    Cardiac Arrhythmia 565
    Angina Pectoris 567
Congenital Affections of the Heart 572

Diseases of the Blood Vessels.

Arteriosclerosis 574
Aneurysm 579
    Aneurysm of the Thoracic Aorta 580
    Aneurysm of the Abdominal Aorta 586
    Aneurysm of the Pulmonary Artery 588

DISEASES OF THE DIGESTIVE SYSTEM.

Diseases of the Mouth.

Stomatitis 590
  Catarrhal Stomatitis 590
  Aphthous Stomatitis 591
  Parasitic Stomatitis 591
  Ulcerative Stomatitis 593
  Mercurial Stomatitis 594
  Syphilitic Stomatitis 594
  Gangrenous Stomatitis 594

Diseases of the Tongue.

Glossitis 597
  Acute Glossitis 597
  Chronic Superficial Glossitis 598

Diseases of the Salivary Glands.

Ptyalism 599
Xerostoma 600
Glass-blower's Mouth 601
Metastatic Parotitis 601

Diseases of the Tonsils.

Acute Tonsillitis 602
  Catarrhal Tonsillitis 602
  Acute Follicular Tonsillitis 603
  Acute Parenchymatous Tonsillitis 603
  Necrotic Tonsillitis 604
Chronic Tonsillitis 605

Diseases of the Pharynx.

Pharyngitis 607
Acute Catarrhal Pharyngitis 607
Chronic Pharyngitis 608
Ulceration of the Pharynx 609
Herpetic Pharyngitis 609
Phlegmonous Pharyngitis 610
Retropharyngeal Abscess 611

Diseases of the Esophagus.

Methods of Examination 614
Esophagitis 615
Acute Esophagitis 615
Spasm of the Esophagus 616
Paralysis of the Esophagus 618
Stricture of the Esophagus 618
Dilatation of the Esophagus 620
Ulcer of the Esophagus 621
Carcinoma of the Esophagus 622

Diseases of the Stomach.

Diagnostic Technique 623
Examination of the Gastric Contents 627
Gastroptosis 633
Gastrectasis 635
Acute Catarrhal Gastritis 639
Acute Toxic Gastritis 642
Phlegmonous Gastritis 644
Chronic Catarrhal Gastritis 644
Gastric Ulceration 653

Cancer of the Stomach 658
Hematemesis 663
Neuroses of the Stomach 665
    Gastralgia 665
    Nervous Dyspepsia 669
    Hyperchlorhydria 672
    Subnormal Gastric Secretion 677
Neuroses of Motility 678
    Increased Peristalsis 678
    Diminished Gastric Peristalsis 679

Diseases of the Intestines.

Diagnostic Technique 681
Enteroptosis 682
Acute Catarrhal Enteritis 683
Chronic Catarrhal Enteritis 688
Cholera Morbus 691
Diarrheas in Children 694
    Acute Dyspeptic Enteritis 694
    Acute Enterocolitis 697
    Cholera Infantum 698
    Celiac Disease 701
    Pseudo-membranous Enteritis 702
Phlegmonous Enteritis 702
Sprue 703
Hemorrhagic Infarct of the Bowel 703
Intestinal Ulceration 704
Intestinal Obstruction 706
Internal Strangulation 706
    Intussusception 707
    Volvulus 708
    Strictures and Morbid Growths 708
    Obstruction from Abnormal Contents or Foreign Bodies 709
    Fecal Obstruction 709
Constipation 714
Carcinoma of the Intestine 722
Hemorrhoids 724
Neuroses of the Intestine 728
    Secretory Disturbances 728
    Sensory Disturbances 728
    Disturbances of Motility 729
Appendicitis 732

Diseases of the Liver and Bile Tract.

Abnormalities of Shape and Position 741
Jaundice or Icterus 742
Icterus Neonatorum 743
Simple Catarrhal Jaundice 743
Acute Infectious Cholecystitis 747
Calculus Cholecystitis 749
Carcinoma of the Gall-bladder and Bile Ducts 753
Stenosis of the Bile Ducts 754
Vascular Affections of the Liver 755
    Passive Hyperemia 756
    Pylethrombosis 757
    Suppurative Pylephlebitis 758
Fatty Liver 759
    Fatty Infiltration 759
    Fatty Metamorphosis 760
Amyloid Liver 762
Perihepatitis 764
Abscess of the Liver 766

- Acute Yellow Atrophy of the Liver 769
- The Liver in Phosphorus Poisoning 771
- Cancer of the Liver 772
- Cirrhosis of the Liver 777

Diseases of the Pancreas.

- Pancreatic Hemorrhage 783
- Acute Pancreatitis 783
- Carcinoma of the Pancreas 786
- Pancreatic Cysts 788
- Pancreatic Calculi 790

Diseases of the Spleen.

- Dislocation of the Spleen 790
- Splenic Hyperemia 791
- Chronic or Passive Hyperemia 791
- New Growths in the Spleen 791
- Amyloid Degeneration of the Spleen 791
- Hemorrhagic Infarct of the Spleen 792
- Splenitis 792

Diseases of the Peritoneum.

- Acute Peritonitis 793
- Chronic Peritonitis 798
- New Growths in the Peritoneum 801
- Ascites 801

Diseases of the Urinary System.

- Diseases of the Kidney 806
- Abnormal Mobility of the Kidney 806
- Circulatory Disorders of the Kidney 808
  - Active Hyperemia 808
  - Passive Hyperemia 810
- Pathologic States of the Urine 813
  - Hematuria 813
  - Hemoglobinuria 815
  - Albuminuria 816
  - Albumosuria 817
  - Indicanuria 817
  - Pyuria 817
  - Chyluria 818
  - Choluria 818
  - Urobilinuria 818
  - Glycosuria 818
  - Acetonuria, Diaceticaciduria, and B-oxybutyricaciduria 819
  - Lithuria 819
  - Oxaluria 821
  - Phosphaturia 822
  - Cystinuria 822
- Uremia 822
- Amyloid Disease of the Kidney 826
- Nephrolithiasis 828
- Acute Nephritis 834
- Acute Interstitial Non-suppurative Nephritis 845
- Chronic Nephritis 846
- Chronic Non-indurative (parenchymatous) Nephritis 847
- Chronic Indurative (Interstitial) Nephritis 858
- Pyelitis 866
- Hydronephrosis 869
- Paranephric Abscess 871
- Cystic Kidney 872
- New Growth of the Kidney 872

Diseases of the Bladder.

Cystitis 875
Stone in the Bladder 879
Neoplasms of the Bladder 879
Vesical Hemorrhage 880
Neuroses of the Bladder 880

DISEASES OF THE NERVOUS SYSTEM.

Diseases of the Peripheral Nerves.

Neuritis 882
Multiple Neuritis 884
Neuromas 887
Neuralgia 888
    Trifacial 889
    Neuralgia of the Neck and Trunk 891
    Neuralgia of the Extremities 892

Diseases of the Cranial Nerves.

The Olfactory Nerve 894
The Retina 894
The Optic Nerve 895
The Optic Tract 895
Diseases of the Motor Nerves of the Eyeball 896
Intraocular Paralysis 896
Diseases of the Fifth Nerve 897
Diseases of the Seventh, or Facial, Nerve 898
Diseases of the Eighth Nerve 900
Diseases of the Glossopharyngeal Nerve 903
Diseases of the Pneumogastric and Bulbar Part of the Accessory Nerve 903
Diseases of the Spinal Portion of the Accessory Nerve 905
Paralysis of the Spinal Accessory Nerve 906
Diseases of the Hypoglossal Nerve 907

Diseases of the Spinal Nerves.

Diseases of the Cervical Plexus 908
Diseases of the Brachial Plexus 908
Diseases of the Lumbar and Sacral Plexuses 909

Diseases of the Spinal Cord.

Spinal Hemorrhage 910
Hemorrhage into the Cord 912
Caisson Disease 914
Spinal Hyperemia 915
Spinal Anemia 916
Spinal Meningitis 916
    Pachymeningitis Externa 916
    Pachymeningitis Interna 917
Acute Spinal Leptomeningitis 919
Chronic Spinal Leptomeningitis 920
Myelitis 921
    Acute Transverse 921
    Chronic 926
Acute Anterior Poliomyelitis 929
Acute Chronic and Senile Paraplegia 932
Landry's Paralysis 933
Tabes Dorsalis 934
Ataxic Paraplegia 942
The Combined Scleroses in Anemias and Cachexias 944

Friedreich's Ataxia 945
Hereditary Spastic Paraplegia 947
Multiple Cerebro-spinal Sclerosis 948
Progressive Muscular Atrophy 951
Amyotrophic Lateral Sclerosis 953
Progressive Bulbar Paralysis 954
Pseudo-muscular Hypertrophy 956
Tumor of the Spinal Cord and Meninges 958
Cavities in the Spinal Cord 960
Syringomyelia 961

Diseases of the Brain and its Membranes.

Pachymeningitis Externa 962
Pachymeningitis Interna 963
Acute Leptomeningitis 963
Tuberculous Meningitis 965
Chronic Hydrocephalus 968
Cerebral Hyperemia 969
Cerebral Anemia 969
Abscess of the Brain 970
Acute Exudative Encephalitis 972
Acute Exudative Encephalitis with Hemorrhage 973
Cerebral Hemorrhage (Hemiplegia) 974
Cerebral Softening 979
Aphasia 982
Cerebral Palsies of Children 983
Tumors of the Brain 985
General Paresis 990

Diseases of Uncertain Pathology.

Epilepsy 994
Hysteria 1004
Neurasthenia 1016
Migraine 1021
Chorea 1024
Huntingdon's Chorea 1032
Choreiform Affections 1033
Paralysis Agitans 1036
Tetany 1038
Fatigue Neuroses 1040
Family Periodic Paralysis 1042
Thomsen's Disease 1043
Exophthalmic Goiter 1043
Acromegalia 1048
Hypertrophic Osteo-arthropathy 1050
Hyperostosis Cranii 1051
Progressive Facial Hemiatrophy 1051
Angioneurotic Edema 1052
Raynaud's Disease 1053
Acroparesthesia 1055
Scleroderma 1055
Localized Hypertrophies 1056
    Symmetrical Lipomatosis 1056
    Unsymmetrical Hypertrophies 1056
    Chronic Hereditary Tropho-edema 1057
Heat-stroke 1057

THE INTOXICATIONS.

Alcoholism 1060
Morphinism 1065
Arsenical Poisoning 1066

Lead Poisoning 1067
Mercurialism 1070
Food Poisoning 1071
    Milk Poisoning 1071
    Meat Poisoning 1072
    Poisoning by Fish or Shell Fish 1072
Grain and Vegetable Poisoning 1073
    Ergotismus 1073
    Maidismus or Pellagra 1073
    Lathyrismus 1073
    Mushroom Poisoning 1074

DISEASES DUE TO ANIMAL PARASITES.

Nematodes.

Ascariasis 1075
Oxyuris Vermicularis 1076
Trichina Spiralis 1076
Uncinariasis (Ankylostomiasis) 1078
Filariasis (Filaria Sanguinis Hominis) 1080
Guinea-worm Disease (Dracontiasis) 1082
Strongyloides Intestinalis 1083
Tricocephalus Dispar 1083

Cestodes or Tape Worms 1083

Trematodes.

Bilharzia Disease 1085
Diastomiasis of the Lung 1086
Diastomiasis of the Liver 1087
Trypanosomiasis 1087
Trypanosomia Fever (Human Trypanosomiasis) 1087
Sleeping Sickness (African Lethargy) 1088
Psorospermiasis 1088
Dhobie Itch 1088
Myiasis 1090

INFECTIOUS DISEASES.

TYPHOID FEVER.

Typhoid fever has been described under various synonyms, such as typhus-abdominalis, enteric fever, pythogenic fever, autumnal fever, etc. It is an acute infectious fever established by the introduction into the intestine and the proliferation of the specific bacillus of Eberth. It is pathologically characterized by the hyperplastic and ulcerative lesions of the intestinal lymph follicles and a secondary toxemia. Clinically it is recognized by its gradual onset, the early appearance of rose spots on the abdomen, a peculiar temperature curve, diarrhea, tympanites, a low grade of delirium, splenic enlargement, a sero-reaction and a marked tendency toward intestinal hemorrhage and a fatal perforation.

The history of this disease may be traced back as far as the writings of Hippocrates and clinical observations pertaining to it were freely made during the seventeenth and eighteenth centuries. Confusion, however, regarding this and typhus fever existed for a long time. In the beginning of the nineteenth century the anatomic lesions were better understood, but it was not until the latter part of the century that the individuality of the disease was clearly established. Credit for this differentiation between typhoid and typhus fever belongs to two American investigators, Gerhard and Pennock. Supplemental study has finally fixed the entity of the disease as we now understand it.

Etiology —*Bacteriology*—The true cause of the disease—the bacillus typhosus—was discovered by Eberth in 1880 and his observations were subsequently confirmed by Koch, Meyer and Friedlaender. The correct morphology of the bacillus and the definite conclusion that this organism is the specific cause of typhoid fever was confirmed, a few years later, by Gaffky, who described the method for isolating it and growing it in pure cultures. The production of the disease experimentally, however, by cultures has not yet been satisfactorily substantiated, though the inoculation of animals with the bacillus has caused fatal results without producing the lesions characteristic of typhoid in man. The fundamental differences between the typhoid bacillus and similar organisms has been clearly established, with particular reference to the bacillus coli communis, while the real infection which creates the fever is, without doubt, secreted by the typhoid bacillus and is called typho-toxin.

The *bacillus of Eberth* is described as a short, thick rod with rounded extremities, three times as long as it is wide, its absolute length being one-third the diameter of a red blood corpuscle. The bacilli may, however, undergo various changes in form, size and arrangement. They are easily stained by the anilin dyes and vacuolations are often developed which are mistaken for spores. In the ulcerating lesions these bacilli may also, by longitudinal application to each other, form filamentous structures which have been described as "pseudo-filaments." In a suitable fluid media both the rods and the pseudo-filaments will exhibit an active spontaneous movement. The bacilli thrive best at the temperature of the body, though they may live and develop rapidly at a lower temperature. They may resist drying for months unless in very thin layers, but the direct rays of the sun will usually destroy them in a few hours. Bouillon cultures are destroyed by carbolic acid—1 to 200—or by corrosive sublimate, 1 to 2500. Their cultivation may be effected upon various culture media such as gelatin, agar, blood-serum and the potato; they will also develop and multiply rapidly in bouillon and milk, thus due consideration must be given when these articles of food are employed.

In the *distribution of the bacilli in the body,* the intestine, the lymph glands, the spleen, the liver, the rose spots, the blood and the bile are the most frequent locations. They may be found in the stools (50 per cent of the cases) the urine, sputum, vomit and the sweat. Suppurating foci and pleural, endocardial or meningeal exudations may also show their presence. *Outside of the body* their existence is somewhat limited on account of the presence of saprophytes. In sterile water they may retain their vitality for weeks and, more than this, they may multiply in this medium. In ice their life may be prolonged for many weeks, while in milk they usually multiply with great rapidity. They also live for a long time in the feces, while the upper layers of the soil may prolong their existence indefinitely. An epidemic of typhoid fever, however, generally implies a repeated or continuous contamination of the media through which they are introduced into the system.

Predisposing Causes.—The geographical prevalence of typhoid fever is confined to the warmer climates to a considerable extent, though it has appeared in all latitudes. The late summer and the early fall contribute the largest proportion of cases, but its appearance is not always confined to these seasons. Hot and dry periods contribute favorably to its epidemic occurrence, which is most liable to develop when the ground water is low, at which time the springs and water sources more naturally drain their supply from contaminated ground. While these seasons and soil conditions seem to favor its prevalence, we must not understand by this that they are essential predisposing influences.

The *age* which is most vulnerable to the disease is between youth and middle life, though we must take note that no age is entirely exempt. After the thirtieth year it is less common, after the fortieth it is an infreqent occurrence, while in old age it is a rarity. *Sex* has even less influence, for, notwithstanding some claims, we must admit that both male and female are equally susceptible under the same exposure when the physical conditions are favorable. It is possible, however, that pregnancy, the puerperal state and lactation diminish the predisposition. Infection in utero is of rare occurrence.

Individual tendency, acquired or inherited, must be given some consideration relating to predisposition, for we must admit that some are more naturally prone to disease than others, while in a few instances we may discover a family weakness which certainly favors typhoid. *Constitutional and social conditions* must be given some weight in the etiological study, for we find that typhoid may favor the well nourished young individual, particularly in case of alcoholic excess. On the other hand, the extensive and serious epidemic usually takes place among that class of people whose physical condition is impaired by poverty and unsanitary surroundings and with whom grief and emotional disturbances are common. The *mode of living* has much to do with its development and, therefore, in the over-crowded quarters where ventilation is poor, food is imperfect and often contaminated, and drainage is impaired, we may naturally expect an epidemic of typhoid to develop. Hence, its occurrence is so frequent in the habitations at a city's outskirts or in camp life. The country towns are not entirely exempt, for the same predisposing conditions may exist here and it often rages in these vicinities with unusual fury. Hospital infection is sometimes a means of its rapid development in ward after ward unless the most painstaking precaution is taken. Diseases which leave a debilitated condition, particularly gastro-intestinal catarrh, will naturally favor the typhoid invasion and propagation. The acute infectious diseases, however, are supposed to prevent it, at least during their febrile stage. It has been asserted that some of these—smallpox and scarlet fever—exert a marked antagonism to typhoid. The observation has also been made that tuberculous patients, at least in the advanced stages, are also protected against its invasion. Where emaciation is marked, in the course of constitutional disease like diabetes, or in connection with malignant neoplasms, the patient is rarely attacked by typhoid. On the other hand, it is probable that some diseases are prone to appear in conjunction with typhoid. This is particularly true of malaria, the symptoms of which are often masked during the typhoid course only to appear when convalescence is established. *Diseases of the nervous system* often furnish a favorable status for typhoid infection. *Immunity* is a matter of uncertainty; some seem to have a congenital protection in this respect, while in others a severe attack of typhoid usually institutes a relative protection from subsequent attacks.

Modes of Conveyance.—In the discovery of a case of typhoid fever we naturally trace its inception back to the bacillus which leaves the body of someone who has suffered with this disease. This is liberated through the agency of the stools, and sometimes by the urine, vomitus and sputum, and it is conveyed to the well person by various media. *Infected drinking water* is the most common mode of conveyance and a majority of the severe epidemics are invariably traced to this source. Wells, storage reservoirs, springs and rivers receive the bacillus either directly from the patient, through the drainage from contaminated cesspools, by infected articles, or indirectly by the introduction of germ-saturated adjacent earth. *Infected milk* is also a carrier of the germ, as it is not only a medium but a nutrient agent as well. The disease will develop when the milk which is used has been contaminated by the dilution with infected water, when the containing vessels have been washed with the same or when a direct introduction occurs from the hands of the milkers. This is the most common source to be investigated in case of typhoid in children. *Other articles of food,* such as salads, celery and fruits, may become carriers of the infection when they are contaminated by the hands of the handlers, by the water which is used to prepare them for the table, or from the infected soil or fertilizer which is used for their growth. Insects which inhabit germ-invaded products may also be carriers of the infection. Infected oysters and raw shell-fish have of late been suspected in this respect, inasmuch as they may fatten upon contaminated sewage or be freshened by polluted water. In like manner we may have cause to fear the use of ice which has been taken from infected ponds; this alarm may nowadays be relieved by the use of manufactured ice. The transmission of the disease by flies and cockroaches has recently been demonstrated.

Contagion or direct transmission may occur by direct contact with the typhoid stools as is instanced by the affliction of nurses who care for the patient, and laundresses who wash the bed and body linen. The *ground soil* is also liable to be a direct agent of infection, particularly if we accept the view of Pettenkofer that this is a means of increasing the potency of the germ. *Transmission through the air,* either in the moist condition or in the form of dust, must be regarded as possible. Hence, sewer gas and sandstorms must be considered as favorable to its introduction, though in all probability few cases result from this cause.

The Forms of Occurrence.—In this connection it is well to keep in mind the fact that the community development of typhoid is somewhat different than in the case of the acute exanthemata. This is particularly true of such pestilences as smallpox, typhus fever, cholera and the plague, which are usually confined to definite portions of the earth in the beginning, and then spread to neighboring or remote countries. Typhoid fever generally occurs sporadically where it is confined to a small group of cases or, in the case of water contamination, it may have an epidemic distribution, though the dissemination maintains a tendency to local limitations. This condition exists because the typhoid germ is not easily transmitted and it must enter the intestine through the media which often pertain to an isolated locality.

Methods of Entrance.—The bacilli obtain admission into the intestine most frequently when swallowed. In order to do this they must survive their passage through the acid secretions of the gastric juice into the intestine where the alkaline juices favor their growth. They primarily invade Peyer's patches and later the mesenteric glands and finally reach the spleen, liver and other organs by means of the circulation. The respiratory organs may favor their introduction into the circulation after a primary lodgment in the tonsils, lungs or pleurae. The bacilli may likewise be inhaled into the pharynx and in this way reach the stomach. In rare instances the germs may reach the kidneys and the cerebro-spinal fluid and thus establish a special clinical symptomatology. It is also claimed that a general infection may exist without localized lesions—a form known as "typhoid septicemia."

Pathology.— The characteristic features of the morbid anatomy which prevails in the primary lesion of Peyer's patches and the solitary glands of the intestine may be considered under four distinct heads. (1) *The stage of infiltration* follows closely after the hyperemia of the intestinal mucous membrane, which is an early sequence of the bacillary invasion. As the diffuse hyperemia and swelling of the intestinal mucous membrane subside, the hyperplasia of Peyer's patches and the solitary glands takes place. This medullary infiltration is not confined alone to these glands, but may invade the lymphoid elements of the mucous membrane as well. These patches may coalesce and form large infiltrated areas in the direction of the long axis of the bowel. The lesions of Peyer's patches are irregularly oval or disc shaped with sharply circumscribed and overhanging edges. At first they are very red, but soon acquire a grayish-red or a grayish-yellow color as exfoliation begins. Infiltration of the solitary follicles gives rise to the formation of round, red or grayish-red elevations, the size of a pea or larger, which project above the mucous membrane. These may assume a fungoid appearance. The involution of this medullary swelling takes place either by resolution, or more commonly by necrosis and sloughing. (2) *The stage of necrosis* follows after the blood vessels have been compressed and obstructed by the cell-infiltration or as the result of localized anemia due to the action of the typhoid bacillus. The necrotic crusts are by degrees cast off from the mucous membrane. While the necrotic process may extend so deep that it involves the serous coat of the intestine it does not usually extend beyond the submucosa. When the necrotic tissue has disappeared the typhoid ulcer remains. (3) *The stage of ulceration* will be characterized by the appearance of ulcers variable in size, shape and depth according to the antecedent infiltration. When the necrosis is superficial they barely penetrate the mucosa, but when it is extensive they extend to the muscular or serous coats of the intestine; their margins are sharp, often irregular and frequently overhanging, while the base is dark red in appearance, though it may assume a slaty discoloration in the later stages. They are usually separated by strips of mucous membrane and a single gland in Peyer's patches may be the seat of several minute ulcers; the entire plaque is rarely implicated. Large ulcers are sometimes found in the vicinity of the ileocecal valve, while other parts of the ileum may present simply hyperemic patches. When the ulcers are situated in the solitary follicles they are smaller, varying in size from one-sixteenth to one-fourth of an inch in diameter. Occasionally the ulcer runs transversely to the long diameter of the bowel and as the result of subsequent cicatrical contraction stenosis may follow. *Hemorrhage* due to erosion of blood vessels is always an alarming feature; it may, however, simply come from the hyperemic edges of the ulcer. Peritonitis is an almost invariable sequence of intestinal perforation. (4) *The healing* process begins with the casting off of the slough. *Granulation tissue* appears on the floor and at the edge of each ulcer and by its gradual extension the necrotic tissue may be replaced. The cicatrix formed by the healing process leaves a smooth though somewhat depressed area in place of the old ulcer. The lymphoid structures at the site of the infiltration are probably permanently destroyed, but the mucous membrane usually regenerates.

Changes in the Mesenteric Glands.—The mesenteric glands are affected similarly and simultaneously with those of the intestine, and the localization of this pathology will be found in the glands of the lower part of the ileum, the cecum and the adjacent portions of the colon. Those of the uppermost part of the small intestine and the lowermost part of the large intestine are rarely involved. The involution of the glands occurs at the same time that it takes place in the intestinal lesions; the swelling gradually decreases, the color becomes paler and they may return to their normal size through the process of absorption. On the contrary a process of softening may develop in multiple areas of the gland and when these coalesce the softened mass may be directly absorbed. A more difficult process of involution occurs when the entire gland undergoes softening. In other instances the gland contents may become inspissated and terminate in calcification, while rupture is an occasional sequence with death from hemorrhage or peritonitis.

Changes in the Spleen.—The spleen becomes enlarged during the onset of the disease, attaining its maximum size at the height of the febrile stage. The enlargement does not assume great proportions, rarely exceeding twice the normal size. In exceptional instances it may show no enlargement and this is probably accounted for by the existence of some previous disease which has thickened its capsule or formed a firm hyperplasia which resists distention. The hypertrophy is caused by its distention with blood and a hyperplasia of its tissue. Following the hyperemia of the early stage the tissue becomes soft and granular. Infarcts may exist as the result of thrombosis or embolism of the splenic arteries and in rare instances abscesses may be found. Still finer alterations in the spleen have been defined by Billroth and Birch-Hirschfeld which are due to the invasion of typhoid bacilli during the febrile stage. These are distributed throughout the organ in innumerable irregular groups with the bacilli so close together that they resemble dark, opaque spots. On rare occasions—particularly in the case of injury—rupture of the spleen has taken place.

Secondary Lesions.—Other organs are affected by the continuance of the typhoid infection. The *liver* becomes hyperemic, firm and somewhat enlarged during the early febrile period and at the height of the disease it is flabbier and lighter in color. A little later in the disease the hepatic structure becomes friable and assumes a grayish-brown color. The cells are at first albuminous and fatty, granular and turbid; later they become swollen and filled with granules and fat drops which finally break up into detritus. The degenerative process is confined to the periphery of the lobule more than the center, but the parenchymatous change may continue to the extent that the organ becomes greatly reduced in size. Wagner discovered small grayish-white nodules in the liver tissue which he defined as lymphoid cells. In rare cases infarction and abscesses have been found. The *biliary* passages frequently suffer from a catarrhal inflammation and occasionally from an ulcerative or diphtheritic process which lead to secondary hepatic abscesses or perforative peritonitis. As typhoid bacilli have been found in the gallbladder the necrotic process is # probably explained by their presence and it may be quite positively inferred that the gall bladder is a source of re-infection.

The *kidneys* likewise are affected by a similar parenchymatous degeneration. At first they are hyperemic and slightly enlarged, but with the progress of the disease they become firmer, lighter in color and the capsule is adherent. Later granular and fatty degeneration of the renal cells take place and in rare instances the lesion may take the form of acute hemorrhagic nephritis. Miliary abscesses may sometimes be found while a catarrhal and diphtheritic inflammation frequently invades the pelvis of the kidney and the bladder as well.

The *lungs* are also subject to pathological change during typhoid fever. *Hypostatic congestion* naturally arises from the prolonged decubitus, and the circulatory depression. *Lobar pneumonia* may follow from the invasion by the diplococcus, the bacillus typhosus or other pathogenic microörganisms. *Hemorrhagic infarction* is of occasional occurrence, and this may terminate in cicatrization or lead to purulent or gangrenous disintegration. *Pleurisy* in the form of a fibrinous deposit may sometimes be detected and when the process becomes septic, pyemia is a possibility. Latent pulmonary tuberculosis may develop during a course of typhoid. The *larynx* and the *pharynx* are each the seat of occasional ulceration with the typhoid bacillus as the probable cause. *Aphthous changes* have also been discovered in these parts while the tonsils may also be affected by the typhoid ulceration. A similar hyperemia and ulceration may extend to the mucosa of the stomach.

The *heart* is an organ which gives rise to much anxiety during the active and convalescent stages. The morbid changes incident to the early period of the fever are not well understood from the fact that few deaths take place during these stages. A certain degree of myocarditis is present in many cases but is not a constant feature. Endocarditis is a rare complication, as is also pericarditis, and, in exceptional cases, fibrinous deposits have been found upon the pericardium. The *blood vessels* are affected in some instances. Obliterating arteritis or a partial arteritis may involve the arteries of the lower extremities and lead to a condition of gangrene. Thrombosis of the femoral vein often result from a phlebitis.

Peritonitis is usually a fatal complication when perforation takes place. When it is not due to perforation it can usually be traced to extension by contiguity from the intestine or mesenteric glands. Parotid abscess may also be a sequela.

The *nervous system* may show a few lesions which are the result of typhoid infection. Meningeal, cortical and deep cerebral hemorrhages are occasionally discovered, while the medulla and the spinal cord are rarely affected. Bulbar paralysis has been a sequela in a few cases on record, while myelitis is liable to result from an embolic focus in the cord. Peripheral neuritis is of frequent occurrence.

The *blood* changes are not marked or numerous. The red corpuscles undergo a slowly progressive reduction during the febrile period, though this reduction is generally slight in young and robust patients. In a relapse the red blood corpuscles show a greater diminution than is seen in the primary attack. If there is a reduction of hemoglobin, it is only relative though it may be augmented during convalescence. Leukocytosis is not present and in many instances actual leukopenia may exist.

Symptoms and Course.—The *incubation period* may vary from eight to fourteen days and in atypical cases it may last even longer. During this time a feeling of lassitude becomes more pronounced from day to day until the more suggestive prodromal symptoms—languor, anorexia, nausea, headache and muscular pains appear. The *general symptomatology* is usually considered in accordance with the signs of the fever which develop from week to week.

Three divisions are, therefore, made: The stage of development, the fastigium or second stage, and the stage of decline.

(1) *The stage of development* usually reckoned from the day the patient takes to his bed, is generally ushered in with a sense of chilliness, though a distinct chill is rarely experienced. *Fever,* gradually increasing in severity, is soon recognized and not infrequently a *nose bleed* suggests the probability of typhoid. Many of the prodomal symptoms now become accentuated. Prostration is extreme, headache is severe and depressing, mental confusion borders upon a mild delirium, the skin is hot and dry, thirst is marked, the tongue is coated, anorexia is pronounced and constipation is a usual condition. The *pulse* is full and slow in proportion to the temperature, the abdomen is slightly distended and the spleen shows some enlargement.

(2) *The fastigium* is that period which usually begins in the second week, though it may appear as early as the fifth day, and continues about two weeks. The *fever* is generally high (103° to 104° F.) with a marked afternoon accentuation, while the symptoms of the first stage are exaggerated. The *pulse* is also accelerated, the mental dullness is increased and there may be decided evidence of delirium, particularly at night. The *tongue* is dry, heavily coated and is protruded with difficulty. *Diarrhea* is a frequent symptom and the spleen gives evidence of increased enlargement. About the eighth day the rose spots may be detected on the abdomen and thighs. During the *third* week, which is the second week of the fastigium, there is an aggravation of all the classical symptoms. The pulse is much higher (110-120), the temperature reaches its highest point (104° to 105° F.) and shows an accentuation of the remittent type. The *delirium* is now liable to take on a violent character, and the *tympanites* is generally more extreme, and many other complicating signs are in evidence. At this time *hypostatic congestion* of the lungs may be present, while there is usually some bronchitis manifested by a slight cough. *Intestinal hemorrhage,* perforation, peritonitis and many other grave complications may present themselves during this stage. It must be kept in mind that all cases may vary from the mild attack to the most severe form and for that reason the symptomatology will not be the same in degree. On this account the second stage may be aborted or prolonged according to the severity of the type and the patient's response to treatment.

(3) *The stage of decline* or defervescence follows closely the second stage in typical cases and usually this may be counted upon somewhere near the twenty-first day. All of the characteristic symptoms now display a tendency to gradually subside. These features, however, may present a wide degree of variance on account of the patient's weakness and the severity of the disease. The *fever* gradually declines from day to day, while all symptoms ameliorate. *New complications* may arise and thus protract the recovery, while a re-infection may develop the typical typhoid characteristics at this time. The treachery of the fever is such that we may reasonably fear the return of the high temperature, tympanites, delirium, hemorrhage or other unfavorable symptoms when a cure seems to be within our reach. A *fatal relapse* may occur at this time if there is any relaxation in dietetic care and treatment. In case of malarial complication the disease may now assume the febrile character of that disease.

Symptoms in Detail.—The *onset* usually comes on gradually and for that reason typhoid is often overlooked. It may appear suddenly with serious nervous manifestations in which headache may be so intense as to suggest cerebro-spinal complications or pronounced facial neuralgia may confuse the diagnosis. Initial pulmonary symptoms resembling pleurisy or pneumonia may likewise obscure the characteristic features while gastro-intestinal symptoms may cause a suspicion of poisoning or appendicitis. The *ambulatory form* is a source of constant error in diagnosis inasmuch as the patient usually keeps about until a temperature of 104° or 105° F., together with the other marked features of typhoid, are suddenly discovered. Cases of this kind frequently terminate fatally soon after the disease is recognized. The *facial aspect* will frequently be very suggestive of the true condition. In the early stages the face is flushed and the eyes are bright, but a little later the expression is listless and apathetic while in the well developed stages it is dull, heavy, and almost besotted. The anemia rarely shows itself until after the run of fever and the prolonged limitation of food, which is essential to treatment.

The *fever* in its regular course rises steadily during the first five or six days, the evening temperature being about a degree or a degree and one-half higher than the morning record.

In the average case it will be as high as 104° or 105° F. during the latter part of the first stage, and it then usually persists during the fastigium with only slight remissions. The *remittent feature* giving to the temperature chart the so-called "step-ladder" appearance, is always a suggestive symptom of typhoid. At this time there may be a difference between the morning and evening temperature of from one to three degrees, while the normal temperature is often reached in the morning. The fever invariably falls by lysis and when the evening temperature has reached no higher degree than 98.6° F. and remained there for several days, we may feel that the patient has attained a normal temperature.

Variations in the typical temperature constantly occur in typhoid and for that reason we may often be disconcerted in our treatment. The characteristic temperature curve can not be counted upon in every case by any means and the different stages which characterize the disease may be decidedly perverted. In some instances there may be a sudden rise, particularly in children, when the attack is ushered in by convulsions. The defervescence may likewise occur in the early stages, while there may sometimes be an inverse type of temperature which is high in the morning and low in the evening. A *sudden fall* of temperature may be expected at any time in case of hemorrhage, and the same is often true when peritonitis ensues, especially as a sequence of perforation. *Hyperpyrexia* is not generally a characteristic typhoid feature, though it may be experienced in exceptional cases, and this will be most frequent just preceding dissolution when the temperature may be as high as 107° or 108° F.

Post-typhoid elevations of temperature may occur after the morning and evening record has been normal for some time. This is termed the *recrudescence* and it is usually without significance, inasmuch as it is unattended by any constitutional symptoms and is of short duration. It is usually the result of some nervous phenomenon or an error of diet. The temperature is sometimes renewed as the result of a septic focus and in these instances septic embolism may be feared. An *afebrile type* of typhoid may exist in rare instances and a subnormal temperature may be found during the convalescence, particularly with children of nervous temperament and anemic diathesis. The *fever of the relapse* is in reality a repetition of the original fever, though it rarely lasts more than a week or two. Chills may develop even during the latter stages of the fever and they invariably indicate a complication of pleurisy, pneumonia, otitis media, prostatitis or some other infection.

The *skin eruption,* which appears usually about the seventh or eighth day, is a characteristic symptom, though it is not always present. It consists of small flattened rose-red papules which are felt by the finger and disappear upon pressure. They are found most frequently in the order mentioned on the abdomen, thorax, back and thighs. They appear in crops which last only a few days, and often leave brownish spots upon their disappearance. A *branny desquamation* may take place in children and, in rare instances, the skin may peel off in large flakes. Other forms of eruption may characterize the skin symptoms of the various stages. For instance, an *erythematous eruption* is sometimes detected in the early part of the fever, *ecchymotic spots* are liable to appear in case of a hemorrhagic diathesis, while *urticaria, purpura, herpes* and the *tâche cérébrale* are exceptional features. *Boils* and in some instances *abscesses* may develop as the result of secondary infection and *peliomata* ("tâche bleuâtres") in the form of little bluish subcuticular spots may be seen, though they are not essentially related to typhoid. *Gangrene* is of rare occurrence on the lower extremities as the result of thrombosis, embolism or obliterating endarteritis. *Profuse sweats,* characterized by Jaccoud as a sudoral form of typhoid, may appear in association with or independent of chills or chilliness. *Edema* of the skin may result from a complicating nephritis; occasionally it may affect a limb following an obstruction of the femoral vein. The hair usually falls out, the nails may become brittle and show transverse ridges indicative of trophic impairment. A peculiar *"musty" odor* may be emitted from the skin, *lines of atrophy* may appear on the skin of the abdomen or on the thighs, while *bedsores* are not uncommon in protracted cases where the dorsal decubitus is prolonged.

The *digestive disturbances* are pronounced features during the course of the fever. *Anorexia* is an early indication of the stomach's inability to care for food and the thirst will demand constant satisfaction. The *tongue,* which is moist and swollen in the early stages, becomes dry and covered with a brownish coating as the fever advances, while at the same time the teeth and lips may be covered with a brownish admixture of food, epithelial debris and micro-organisms called *sordes*. This condition may often be obviated by a watchful cleanliness of the mouth. *Parotitis* may exist as the fever begins to subside and the *pharynx* may be the seat of a slight catarrhal invasion. The *tonsils* are the seat of a peculiar ulcerative process which is supposed to result from the specific action of the bacillus. *Thrush* is often a sequence of this. The *gastric symptoms* are variable, though nausea and vomiting may be experienced at times. *Hematemesis* is a rare symptom. The *intestinal symptoms* are prominent features inasmuch as initial lesions are there. Constipation is typical of the early stage and may continue through the course of the fever. *Diarrhea* may occur at the beginning and it may be a persistent feature for some time; it is most severe and protracted when there is an extreme catarrhal condition of the large intestine or when the ulcerative process is limited to the colon. *Involuntary discharges* from the bowel may take place and this occurrence is usually an unfavorable symptom. The stools in the ordinary case have a consistency and color of pea soup; they are offensive in odor, alkaline in reaction and when allowed to stand, separate into a thin and serous layer containing albumin and salts and a lower stratum consisting of epithelial debris, remnants of undigested food and numerous crystals of triple phosphates. Microscopic investigation may reveal the presence of some blood, necrotic crusts from Peyers patches, with occasional portions of bowel tissue. The typhoid bacilli may be found in the dejecta at the end of the first or during the middle of the second week. *Tympanites* due to decomposition and fermentation of food, especially in the colon, may be very troublesome and is usually more marked when diarrhea is present. It is not always a marked feature of the disease and will usually be limited by the careful regulation of the diet and the use of intestinal antiseptics. Constipation may favor it by the retention of decomposing particles of food. Abdominal pain and tenderness are not usually extreme unless there is extensive ulceration, or other complicating conditions, like peritonitis or appendicitis which produce it. The absence of these symptoms, however, do not indicate necessarily a mild lesion. Pressure over the right iliac fossa may elicit some pain and a gurgling noise is occasionally detected by pressure over the ileo-cecal region.

Intestinal hemorrhage is said to occur in from three to eight per cent of all cases, though this percentage is probably reduced under the modern methods of treatment. It generally appears about the third week as the result of erosion of the intestinal blood-vessels during the ulcerative process. Slight hemorrhage may also take place during the earlier stages in patients of the hemorrhagic diathesis or it may be the result of an intense hyperemia. Under all circumstances hemorrhage, even when slight, should be regarded as a grave symptom, though it does not necessarily indicate a fatal result. Signs of collapse are in evidence and the temperature may drop eight or ten degrees in a few hours. The blood may be profuse or may be recognized only by the tarry appearance of the stool; though, in some instances, a fatal collapse may take place without any sign of hemorrhage from the bowel.

Perforation is a condition always to be feared inasmuch as it invariably produces a fatal diffuse peritonitis. It may occur at any time during the fever, but it is most common between the second and fourth weeks. Hemorrhage is a presumptive evidence, though it may occur without premonitory signs even in mild cases. Males are said to be affected more than females and age must have some influence inasmuch as children are rarely afflicted. *Pain* which is sudden, sharp, paroxysmal and increasing in severity, is the most characteristic symptom. It may be diffuse though frequently located in the hypogastric region to the right of the median line, and it may radiate toward the pubes and be attended with pronounced vesical irritability. *Sudden collapse* usually follows the pain, the temperature falls, the pulse increases in rapidity and there is a marked tendency to sweating. Palpation will reveal a general tenderness in the hypogastric region or this may be localized so that it is observed only on deep pressure. Muscular rigidity and increased tension will be evidenced while a spasm may follow an attempt to palpate. Rapid respiration with a rapid, thready pulse, subnormal temperature, pallor, a pinched facial expression, hiccough and vomiting, are the symptoms of collapse. A leukocytosis, though not constant, may be a valuable early sign which should suggest prompt surgical interference. The *abdominal signs* of perforation, although not constant, should be carefully studied. The limitation of abdominal respiratory movement, the increasing distention, the augmented pain on pressure and the muscular spasm are clinical points of value. It must be remembered, however, that perforation and peritonitis may exist when the abdomen is flat or scaphoid. Percussion may reveal a flat note in the flanks as the result of the exudate. Peristalsis may be diminished, while auscultatory percussion will sometimes show the presence of air free in the peritoneal cavity. The liver flatness in the nipple line may be obliterated by excessive tympany, while the rapid obliteration of the liver flatness, in case of a slightly distended abdomen, will be a suggestive symptom. Generalized peritonitis may succeed the circumscribed variety as the result of direct inflammatory extension or it may follow from the rupture of a suppurating mesenteric gland.

The *spleen* generally shows evidence of enlargement during or before the fastigium; a gradual increase takes place until the fourth or fifth week when there is tendency toward subsidence. The enlargement may disappear rapidly after a copious intestinal hemorrhage. In some cases it may not be demonstrated at any time during the fever. Softening of this organ may follow the presence of suppurative infarcts while cases of splenic rupture have been reported. The *liver* likewise is subject to enlargement during the course of typhoid fever. Jaundice may evidence this when there is a catarrhal invasion of the ducts or in case of hepatic abscess or gall stones. Cholecystitis may follow from the invasion of the ducts by the typhoid bacillus. The suppurative lesions are liable to result in perforation with secondary peritonitis while suppurative pylephlebitis may follow a perforation of the appendix.

Respiratory symptoms attending typhoid fever will be recognized in many forms. *Epistaxis* is an early occurrence in many cases and it is occasionally profuse when the disease is more advanced. It has grave significance when occurring during the fastigium. *Laryngitis* is a rare complication of slight significance, but *bronchitis* is often a marked feature of the disease. This will be characterized by the presence of numerous sibilant râles while a more severe bronchitis may follow after the patient has remained in the dorsal decubitus. In advanced age emphysematous symptoms may enhance the severity of the bronchitic complication. *Pneumonia* is also liable to be a serious complication during the later stages of typhoid and the condition may be easily overlooked on account of the absence of its cardinal symptoms. *Hypostatic congestion of the lungs,* as has been previously mentioned, may be a sequence of the enfeebled circulation during any period of typhoid. This is usually bilateral and is associated with a considerable degree of pulmonary edema.

The *circulatory disturbances* are not marked until the asthenic stage is reached. In severe types of the fever the first heart sound may be weakened as a result of myocardial involvement. It is often the cause of sudden circulatory failure. Endocarditis and pericarditis are of rare occurrence. The *pulse* is rapid, but it is not accelerated in proportion to the temperature until myocardial weakness appears. The high pulse, therefore—120-130—is suggestive of a serious condition inasmuch as the average pulse is not usually much above one hundred to one hundred and five. It is, of course, enhanced during the later stages of debility or from neurotic or anemic causes. The dicrotic feature will indicate the lowered arterial tension and bradycardia, due to toxemia, may exist for some time after recovery. Thrombosis of the femoral veins is of common occurrence, the left being more frequently involved than the right. Gangrene of the foot may follow an obstruction of the venous or arterial circulation.

The *blood changes* in typhoid constitute an important phase of the symptomatology. During the first two weeks there may be only a slight variation from the normal, and it is even claimed by Hayein that profuse sweats or a copious diarrhea may cause an increase in the corpuscles. About the third week, however, there is a decrease in the number of red corpuscles which may become greater as convalescence or asthenia take place. The hemoglobin suffers a greater relative reduction than the red corpuscles. The white corpuscles remain slightly below the health standard until the convalescing period when a moderate leukopenia may exist. A leukocytosis may be discovered in case of hemorrhage or perforation or when the disease is complicated by peritonitis, pneumonia, pleurisy, phlebitis or any other infective process. A cold bath is said to increase the number of colorless corpuscles.

The *nervous system* is the seat of much disturbance and this will be manifested by various symptoms. *Headache,* which is an early feature, may extend into the cervical region and when it is accompanied by such symptoms as rigidity, retraction of the head, photophobia and muscular twitchings, it may give rise to a suspicion of meningitis, which, however, is of rare occurrence in typhoid. Wakefulness, restlessness and vertigo may be bothersome symptoms at times, but mental dullness is usually the characteristic feature. *Delirium* is a common indication of toxemic irritation. It is generally of a mild character and is enhanced at night by mild delusions. The patient more frequently lies in a somnolent state with a muttering delirium which is the typical typhomania. This may give way to a gradually developing coma; the latter, in serious cases, may be a sudden development. A wild delirium occasionally develops and in such a state it is often necessary to restrain the patient in bed. The worst form of delirium is that which is experienced in alcoholic cases and this requires the administration of powerful sedatives to keep the patient quiet. Sometimes the delirium may assume the hysterical type. Motor nerve disturbance may often be recognized in conjunction with the delirium; this will frequently be confined to facial twitchings, intentional tremors or a peculiar twitching of the wrists and fingers which is called subsultus tendinum. The *tongue* is generally protruded with difficulty, while the lips and lower jaw are often in a state of constant tremor. The reflexes are increased except in case of coma when they are generally abolished. The cause of these motor perversions is, no doubt, due to the action of the toxins upon the nerve cells. *Nervous sequelae* often follow a prolonged run of typhoid and chief of these is the paralysis of one or more muscle groups, which often is the result of a post-typhoid neuritis. *Hemiplegia* is an occasional sequence when cerebral hemorrhage or localized encephalitis attend the disease. *Aphasia, mental disturbances* and cranial neuralgias are rare complications which may follow a prolonged course of the fever. The *typhoid spine* has also been described as a condition incident to an acute inflammation of one or more vertebrae when the fever has been unusually protracted. Many neurasthenic symptoms which the patient complains of are, no doubt, traceable to this infection long after recovery is apparent.

The *urinary changes* will vary according to the severity of the typhoid attack. During the first two or three weeks the amount of urine is decreased, while it is not uncommon, particularly when nervous symptoms are marked, for a polyuria to exist. The specific gravity is high and the reaction is decidedly acid until the later stages when the former declines and a neutral or alkaline reaction may appear. Urea and uric acid are at first increased while the reverse is true as convalescence begins. The chlorids are always diminished with the febrile development but they invariably increase when the decline occurs. *Febrile albuminuria* is a common complication in a large percentage of cases and deserves careful consideration in the treatment. In addition to this we may expect the presence of blood and casts should an acute nephritis develop, while uremic symptoms and a chronic nephritis are constant features to be feared in protracted cases. When the typhoid terminates in a normal or an early convalescence these complications are generally rare. A *post-typhoidal pyelitis,* invading the pelvis and calices of the kidney, often gives rise to a discharge of pus and blood in the urine. The diazo-reaction will be a diagnostic help in kidney complications, though we must remember this may take place in phthisis, meningitis, pneumonia and other infections. *Cystitis* not rarely due to the typhoid bacillus as evidenced by its presence in pure culture in the urine may be a complication; it may, however, be due to other pyogenic bacteria. Diseases of the genito-urinary tract are dependent to a great extent upon local infections which have preceded the typhoid fever.

The *joints* are subject to arthritis during the convalescing period of typhoid. The infection may be purely typhoid or more commonly it is either rheumatic or pyogenic. In the first instance the arthritis is monarticular, but it is polyarticular when either of the latter two infectious agents is the cause. The *muscles* may undergo a hyaline degeneration and may be the seat of hemorrhage or abscess. The *bones* and periosteum are more frequently disposed to typhoidal lesions and, therefore, periostitis with secondary necrosis and osteomyelitis may occasionally prolong the convalescence or appear as a sequela.

Post-typhoid septicemia and pyemia may be observed in protracted cases after the deferescence. These signs will be recognized by the profound intoxication with slight fever and septic sweats. Again recurring chills, often of great severity, may be followed by more pronounced flights of temperature. The pyemic condition will often be evidenced by the presence of furuncles and multiple abscesses and the associate irregular fever with leukocytosis. The suppurative process may likewise be established by a femoral thrombus; it may also come from a splenic infarct, a perinephritic or perirectal abscess, a suppurative arthritis and other sources of pus development.

Varieties of Typhoid.—The symptomatology of typhoid fever is subject to such variations as may arise from an exaggeration or modification of its typical symptoms. Again the complications may be so prominent that we employ such compound terms as nephro-typhoid, pneumo-typhoid, pleuro-typhoid, etc. These are, however, unfortunate terms inasmuch as they do not represent distinct varieties of typhoid. There are, however, varieties which need some consideration.

The *mild* and *abortive forms* may receive first attention. The former is not so intense in character, though the symptoms are often typical and the onset may be sudden and severe. The temperature does not run so high and the gradual afternoon accentuation is not usually apparent. The spleen is generally enlarged, the rose spots are often in evidence and the gastro-intestinal disturbance may be present, but the fever does not last more than ten or fifteen days. The abortive form may have a sudden onset with all the typical succeeding symptoms, but at the end of the first week or early in the second the fever disappears with profuse sweating. Convalescence usually follows without further complications, though a severe relapse may take place. The *grave form* of typhoid will be suggested by a profound systemic intoxication, by the high fever, the severe nervous symptoms and the marked prostration. In this class we may include those cases with cerebro-spinal, nephritic and pneumonic complications. The *latent* or *ambulatory form,* which has been referred to before, will be recognized in those patients who keep about during the early stages of the fever and only succumb to its ravages when the serious symptoms appear. Delirium, high temperature, tympanites, hemorrhage and perforation, as the result of dietetic errors and lack of care, are liable to characterize the final stage which often terminates in sudden death. The *afebrile form* is of rare occurrence. *Hemorrhagic forms* of typhoid are also rarely observed, though they are occasionally typified by an hemorrhagic onset or the supervention of the same during the febrile course. These are not necessarily fatal, though they are extremely serious.

Typhoid in children is not very common, though it may occur under ten years of age and it has been reported in infants. The onset is generally abrupt, but many of the prodromal symptoms are wanting. The temperature is characterized by a gradual rise, while bronchial catarrh and an exaggeration of the nervous symptoms are prominent features. The rose spots are not always present and the abdominal symptoms are mild. Fatal hemorrhage and perforation are exceptions, though the sequelae are often serious, but ordinarily the mortality is not high. It is claimed that the fever may exist in the fetus, the bacillus passing through the placenta to the child, causing thereby a typhoid septicemia. If this is true, it is a proof that pregnancy does not afford immunity. *Typhoid in the aged* is not common and when it does occur the mortality is high inasmuch as pneumonic and cardiac complications are common and severe.

Relapses of Typhoid..—Relapses in typhoid fever are a constant menace. An intercurrent relapse may develop before the temperature becomes normal, but more frequently it appears during or some time after the convalescence. The early theory regarding a relapse held that it was due to dietetic error before the intestinal lesions had healed. This, no doubt, has a semblance of truth, but the real cause arises from re-infection. For a time it was supposed that this took place in the large intestine as the result of the passage of the infected sloughs over healthy lymphoid follicles. The more probable theory is that the gall-bladder contains numerous bacilli which may, at varying periods after the primary infection, be discharged into the bowel or circulation and cause a re-infection. This explains the influence of a dietetic error which stimulates the action of the liver and causes the bacilli to be poured with bile into the intestine. In this way they re-enter the circulation and cause a return of the primary typhoid symptoms. The relapsing attack is generally less severe, shorter in duration and the cardinal symptoms, aside from the fever, are not always marked. The onset may be sudden and the rigors severe. There may be a number of relapses and they are more likely to occur in young patients. Great care should be exercised to discriminate between a relapse and a recrudescence or spurious relapse. The prognosis should also be guarded inasmuch as a relapse which follows a mild attack of typhoid may present graver symptoms than the latter.

Diagnosis.—The diagnosis of typhoid fever is not in every instance an easy task. For that reason it is always best to regard suspicious symptoms with consideration until positive signs are disclosed. It is by all means safer to consider a fever as typhoid than to fail to recognize a mild or abortive form. A diagnosis should always be postponed when the prodromal symptoms are inconclusive and the patient should be placed in bed and put on a limited diet while the symptoms are carefully watched from day to day. The presence of epistaxis, diarrhea and such objective symptoms as splenic enlargement, tympanites, tenderness in the right ileo-cecal region, which usually follow the prodromal symptoms, together with a pulse which is slow in proportion to the temperature and often dicrotic, will help to make our diagnosis. When the rose spots are found and the temperature shows its peculiar curve, we may begin to appreciate and understand the various symptomatological features which have been previously mentioned.

The *sero-reaction,* which is frequently found as soon as the second week, and sometimes earlier, will be of great diagnostic help. It is generally known as the Widal reaction inasmuch as this investigator demonstrated the specific value of this procedure in clinical medicine. The serum theory depends upon the fact that the diluted serum of a patient afflicted with typhoid fever will cause the bacilli to lose their motility and become aggregated in clumps. To obtain this will require the services of one who is skillful in the required technique. The standard dilution of 1-50 and a time limit of one hour are important requirements. After the serum is mixed with a fresh bouillon culture of the typhoid-bacillus so that the required dilution is obtained, a hanging-drop preparation of the mixture is made; if there is a positive reaction the bacilli will, in an hour's time, lose their motility and collect in clumps. Wyatt-Johnston suggests the following method: "The blood is taken on a clean glass slide from a needle-prick; a watery solution of this dried blood specimen is added to a loop of the bouillon culture of typhoid bacilli on a clean cover glass and the microscopic evidence of the disappearing motility and the clumping are then obtained." While this is a fairly positive evidence of typhoid, it is not always an easy matter to obtain it and many times it can not be applied. Interfering conditions like tuberculosis and pneumonia may make the reaction negative. In some cases with typical symptoms the reaction may not appear at any time.

The bacilli may be isolated from the stools, the urine and from the rose spots. Aside from these specific disclosures we must at all times be guided by a careful physical examination and the consideration of the typical symptoms. In the presence of an epidemic our diagnosis will be less in doubt.

Ehrlich's diazo-reaction mentioned under diseases of the urinary system, is obtained as follows: One part of a solution of sodium nitrite (0.5 per cent solution) is mixed with forty parts of a solution consisting of sulfanilic acid two grams, hydrochloric acid one hundred and fifty c.c. and distilled water one thousand c.c. To this an equal volume of urine is added, and the test-tube thoroughly shaken. Now superpoise a small amount of ammonium hydrate and if the reaction is positive a ruby or cherry red ring occurs at the line of contact. The value of the above has already been discussed.

Differential Diagnosis.—Since it is possible to apply the sero-reaction in so many cases, there is not so much danger of confusing typhoid with other febrile diseases. Still a confusion may exist in many instances. *Typhus fever* has a shorter course, terminates abruptly by crisis, and has no Widal reaction. Its symptoms portray a more sudden onset, the stupor is more pronounced, the conjunctivæ are injected and it is further characterized by the appearance on the third to fifth day of maculæ which are soon transformed into petechiæ. It has also more marked nervous manifestations and death is liable to ensue at the end of the second week. *Acute miliary tuberculosis* will have a different temperature curve, the pulse and respirations are more rapid, the rose spots are not present and the Widal reaction will not be detected. The cough, hemoptysis, peculiar cachexia and the absence of abdominal symptoms will generally overcome the diagnostic doubt. *Malarial fever* will often cause much doubt in the atypical typhoids, but the blood examination will invariably reveal the malarial organisms. *Relapsing fever* has more of an abrupt onset and the rigors and fever are more pronounced at first. The duration, however, is shorter, the eruption, the sero-reaction, the nervous symptoms and the temperature curve are usually absent. The termination is by crisis and the presence of the spirilla is a diagnostic distinction. *Meningitis* will be differentiated particularly by the accentuation of nervous symptoms, the headache and the character of the delirium. The muscular rigidity which takes place before the effusion, the hyperesthesia, the exaggerated reflexes, Kernig's sign, the restlessness, vomiting and lower temperature record will establish a differential symptomatology. Lumbar puncture in meningitis will generally give a positive microscopic diagnosis. Tubercular meningitis is recognized in younger patients, the Widal reaction is absent and the other tuberculous manifestations are usually found. *Pneumonia* in its typical course rarely simulates typhoid, but there is an atypical form of pneumonia which resembles typhoid so closely that the name "typhoid-pneumonia" has been accepted (see under pneumonia). *Catarrhal enteritis* in children may often resemble a typhoid fever in its earlier stages, but here the symptoms pertain entirely to the gastro-intestinal tract and, aside from the fever, the other features of typhoid are absent. *Salpingitis and appendicitis* will often cause the greatest amount of doubt in diagnostic differentiation with typhoid. These, however, may be differentiated by a careful consideration of their respective physical findings.

Prognosis.—The prognosis in typhoid fever will depend upon the gravity of the case, the character of the epidemic and the physical condition of the patient. The average mortality record is certainly not so high under the recent method of treatment as it has been in the past. Much depends upon the early diagnosis and the regulation of the diet. Many patients have reached the third week of the disease before they enter a hospial or call a physician and this, of course, militates against a favorable prognosis. High fever, delirium, with signs of marked toxemia, meteorism, hemorrhage and the evidence of perforation are always danger signs. Nervous complications, particularly the tremor which attends a low muttering delirium and the involuntary action of the sphincters are always to be considered as unfavorable. The ambulatory form of the fever usually terminates fatally. Sudden death occasionally occurs when the symptoms seem to indicate improvement, and the immediate cause is not always discovered. In all probability such a termination is due to pulmonary embolism or myocarditis which ends in a sudden cardiac failure. A recurrence rarely takes place after a typical attack and, therefore, such patients are seemingly immune.

Treatment.—Prophylaxis is essentially a part of the treatment as preventive therapeutics is of first consideration under this subject. This pertains not alone to efficient drainage in the vicinity where typhoid is epidemic, but it refers most emphatically to the proper isolation of the patient and the careful disinfection of all excretions. A vessel which can be easily disinfected within and without should be used to hold the excreta, the urine, vomitus and sputum. These should be disinfected with a solution of chlorid of lime—six ounces to a gallon of water, bichlorid of mercury—1:500, or a five per cent solution of carbolic acid and allowed to stand for an hour or more before they are thrown into the closet. Dishes, bedpans, urinals and rectal tubes must be isolated and boiled; thermometers must be kept in a bichlorid solution (1:1000); the soiled linen must be soaked in carbolic water (1:20) for two or three hours before they are sent to the laundry; blankets, mattresses and pillows must be sterilized by steam or formaldehyd, while the nurse's hands must be carefully scrubbed and washed in bichlorid solution after handling the patient. Carelessness in these details is too frequently tolerated by physicians. Perfect cleanliness and asepsis should be maintained about the patient, bed and room, and to successfully accomplish this, absolute isolation is necessary. The water and milk which are given to the patient and used by the household should be thoroughly boiled. Preventive antityphoid-inoculation has come into favor recently and the best evidence of its efficiency was obtained during the South African war where it reduced the occurrence in those inoculated to less than 1 per cent. This method of prophylaxis is still in its infancy, but it will no doubt be a decided feature in the future treatment of this disease.

Treatment of the Attack.—The *general management* of a case of typhoid fever must contemplate, first of all, the necessity of keeping the patient absolutely quiet in bed. This requirement is of such vital concern in the care of all acute diseases that it is naturally the first step in the treatment. It is however, of paramount importance in typhoid fever, inasmuch as the nervous system then is prone to unusual excitement. Quiet should be enforced not alone so far as the patient is concerned, but it should be applied with equal force to the surroundings. A trained nurse should always be in charge since the essential scientific care can not be administered by sympathetic relatives or friends. The patient should be alone with the nurse except at stated periods and the room should be kept well ventilated, with an equable temperature and with as cheerful surroundings as possible. The bed should be protected by a rubber sheet so that the baths may be given without disturbance. The patient and the linen should always be clean, the mouth should be frequently cleansed and the greatest care should be exercised to avoid the development of bedsores. Attention to the welfare and comfort of the patient which result from skillful nursing favor the absence of complications and a speedy convalescence.

The *diet* is essentially of first consideration and, though various theories prevail in this respect, it is certainly agreed that it should be limited in quantity, restricted to semi-solids and administered at regular intervals. The majority of patients will subsist safely on a minimum amount of milk frequently administered. During the first forty-eight hours and possibly for several days, it is often sufficient to give nothing but sterile water ad libitum. In the early stage it is my plan to give every two or three hours two to four ounces of milk, diluted with an equal part of water, both of which are to be sterilized. If the fever is not particularly high and if there is evidence of undue weakness, it is better to increase the amount so that at least three pints are taken daily, and if this quantity is tolerated, it may be well to cease diluting it. When a diarrhea persists, the milk should be boiled. The tolerance of milk as a food will be better understood by carefully watching the bowel movements; when curds and fat globules are found, it is evident either that milk is not an acceptable food or that further dilution and decrease in quantity is necessary. In such cases lime water or a little brandy may be added to each feeding or the peptonizing process may be instituted; if then it is not digested, whey, sour milk, buttermilk, Matzoon or Kumiss should be substituted. It is my belief that, if these measures are carefully followed out, few patients will reject this diet unless they have a particular idiosyncrasy against it. Egg-albumen, made by straining the whites of eggs through a cloth and mixing with them an equal amount of water, or eggs beaten up in milk, may be used when milk is not acceptable; the addition of a little lemon, vanilla or whiskey will often make this more palatable. A well strained barley gruel, or other bland gruels, are frequently serviceable. Meat-juices, chicken, mutton or beef broths, at first well diluted, may be substituted or alternated with the milk when the latter disturbs or does not suffice; beef peptonoids are highly recommended, but the disagreeable taste is often objectionable. Broths are always to be used with greatest caution and in case temperature and other cardinal symptoms are noticed after their administration there should be a prompt return to the milk diet. It must be understood at all times as a good principle of practice not to disturb a patient during a natural sleep, to administer food. To a certain extent this theory should apply when delirium is acute or mental excitement prevails. When a marked improvement is noticed or the strength begins to fail, more substantial nourishment will be demanded. In such instances scraped beef, soft toast, custards, gelatins, etc., may be well borne. Diarrhea is usually an indication that the nourishment is causing intestinal disturbance. In case the oral feeding is not sufficient to combat the failing strength or the stomach refuses food, it is necessary to resort to rectal alimentation. For this three or four ounces of peptonized milk or a combination of this and a little white of eggs may be employed every three or four hours. Other forms of nutrient enemata may be demanded according to the complications of the case. In the early period of convalescence it is safe to give a soft boiled egg, gelatin, custards or some well cooked rice, scraped beef, etc., at long intervals. If no disturbance is noticed from this innovation, gradual resumption of a light diet may be instituted. It must be recognized, however, that this innovation is the greatest danger of our treatment. It is by all means best to delay the return to solid food until there are positive signs of a safe convalescence; even then this end must be attained gradually and as long as the patient is properly sustained by a soft food the regular diet must not be resumed until the danger point is positively passed. *Water* should be given freely during all the stages and in many instances the patient may tolerate barley water, cocoa and perhaps a little lemon or orange juice.

Stimulants are dangerous things to deal with, but in many instances they are absolutely required. Cardiac enfeeblement and general exhaustion may demand the use of whiskey in limited quantity, particularly during the middle or later stages of the disease. It will likewise combat the typhoid septicemia, and to a certain extent control the delirium, tremor and other nervous symptoms. It will be indicated more frequently when the patient is an alcoholic or when sudden collapse is feared. Heart stimulation should not be employed in this or any disease until it is absolutely required. When the pulse is persistently high (110-130), it is a wise procedure to resort to strychnia (gr. 1-60 to 1-30) by mouth or hypodermatically, three or four times daily, but to gradually discontinue its use when the heart attains a more normal condition. Digitalis, strophanthus or the combination of strychnia and digitalin are often more serviceable. The hypodermatic use of sterile camphorated oil or five grains of camphor in sterile almond oil often gives good results in case of sudden and extreme cardiac enfeeblement. I think it is safe to say that stimulation—particularly in children—is rarely required.

Hydrotherapy must be regarded as one of the most important features in the treatment of typhoid fever. With this and the limitations of diet, together with good nursing, it is possible to carry a case to a safe convalescence without the aid of medicine. It is, therefore, essential to give due weight to this phase of treatment throughout the entire course of the disease. Hydrotherapy is such a valuable and at the same time such an abused expedient that it requires some thoughtful consideration. A variety of opinions as to the method of its use still confuse the profession. Too many employ it indiscriminately, and some methods seem barbarous in certain instances at least. That the cold bath, as employed by Brand, may be safely and efficiently used in some cases I will not deny, but to employ it in all cases I truly believe is a mistake. To put some delicate patients into the cold water is a shock which can not be endured, to say nothing about the irritation of the nervous system. I am too mindful of the fatal reactions in a few experiences of this kind to permit me to adopt it as a universal method. The cold pack is a much more merciful process, and I believe it is fully as effective. In the average case I prefer the cool sponge systematically employed, and in some instances warm or tepid water is preferable. My usual plan is to give the cool sponge in this way: The nurse is to bathe the patient for ten minutes, then allow the drying process to take place while the patient rests for ten minutes. This is then repeated twice in an hour, with an equal rest period following each time. Thus the patient is sponged for one-half hour intermittently and also rests for the same length of time in one hour's attention. Following this, absolute rest is enforced for several hours if possible. The sponge is repeated in accordance with the temperature. At least one sponging of this character must be given each day. In my experience no better method than this has been found. Ironing the patient with a piece of ice over a sheet is another expedient which gives favorable results. Aside from the disagreeable features of the Brand system of treatment, there are several conditions which absolutely contra-indicate the cold bath; these are intestinal hemorrhage, peritonitis, and extreme cardiac weakness.

Intestinal antiseptics have been employed for some time with the hope of destroying or inhibiting the germ growth. It would seem natural that the principle of antisepsis ought to apply in typhoid fever. Unfortunately we have not as yet any satisfactory proof that it does. The bacilli accomplish their organic work long before the patient consults the physician, and while we admit the cause, we never know how long the latent influence has existed. The symptoms which present themselves for treatment are mainly the result of toxins and probably a mixed infection. Thus it would seem necessary in order to apply the correct principle of antisepsis, that we must gain entrance to the intestine, without any antidotal influence, with our destroying agent as soon as the germ develops. How we may get the desired effect by any antiseptic which acts through the blood or otherwise has not yet been discovered. Notwithstanding this apparently discouraging prospect, I am still of the belief that intestinal antiseptics are a valuable adjuvant in treatment. If it is impossible to destroy the bacillus, we may possibly neutralize or at least inhibit the toxin development by this means. If such a treatment does not reach the specific cause, it certainly has a tendency to arrest intestinal fermentation and destroy the intestinal bacteria which favor the fever. While I am not able to speak with scientific positiveness on this subject, I have certainly satisfied myself of the practical value of this adjuvant. In my hospital and private practice, where I have had the privilege of observing a great many cases, I have tested this principle in a clinical way for several years. I am so convinced of its efficacy that I do not hesitate to employ an intestinal antiseptic from the very beginning.

The remedies employed for this antisepsis are numerous. Many have been discarded by the authorities who first advised them. Since the experiments with chlorin water years ago many agents have been resorted to. Compounds of carbolic acid and iodin, beta naphthol, salol, thymol, turpentine, benzosol, boric acid, etc., have been carefully tested. Even the idea of elimination by the use of calomel relates to this theory for by the excessive introduction of bile into the intestine the antiseptic theory is applied. While we may justly oppose the mercurial irritation for its unfortunate reactions, we may at least admit that milder measures of that kind are essential to the proper treatment. In my practice I have received the most efficient results from the use of *methylene blue.* I gave this in the beginning in the first decimal potency, four tablets three or four times daily. Recent experience has taught me that this is not a sufficient dose, and I have employed chocolate coated pills of one to three grains four times daily. Since I have used this I have lost few cases. This, no doubt, will appear to be a boastful statement, but it is evidenced by the result of many cases covering several years of experiment with this agent. I no longer dread the treatment of typhoid as I did in the past. With its use the tympanites, the fever and the delirium are minimized. I rarely now have a case of hemorrhage, and the terrible subsultus and general nervous excitement do not attend. I am therefore rarely obliged to use restraint, and I enter upon the treatment with less fear and dread. Though I can not prove my claim as an absolute verification of the antiseptic theory, I am still sufficiently satisfied that the generation of toxins in the bowel and their absorption is much reduced by the employment of this agent and hence its clinical value. *Guaiacol* has of late been employed as an internal antiseptic, but its efficiency is more pronounced as an antipyretic. For this purpose ten to fifteen minims are rubbed into the groin when the afternoon temperature is at its highest. This dosage is liable to produce untoward effects and therefore it has been my custom to employ only five minims. I am pleased to state that, in a limited experience with this agent, the results have been very favorable, though I am not satisfied that it should supplant the hydrotherapeutic measures. To gain success with this agent it is necessary to occupy at least one-half hour to rub in the medication.

The care of the bowels will naturally call attention to the necessity of using enemas. There are many who still believe in not disturbing the bowels in this way, except for obstinate constipation, which they claim is rare. In controversion of this idea I am willing to state that a fair average of my cases are constipated and often need medical and mechanical help. The enema is, in such cases, extremely serviceable, but I believe it has a more general value in the sense of intestinal irrigation and absorption. The more water we get into the system of the typhoid patients the better it is for them. In the use of the enema some of the fluid will be absorbed, and its cleansing effect is of course admitted. My preference is for the high normal salt enema, and I use if possible a quart and have it retained. The beneficial effect is invariably shown by the reduction of the temperature at least a degree in a short time. If the temperature remains above 103° F., I have it repeated and in severe cases it may be used several times daily. Involuntary bowel movements would contraindicate it. The average case in my practice gets it every morning. The systemic effect obtained from the regular use of the high normal salt enema is apparent by the absence of extreme tympanites and delirium. Of course we all recognize the benefit obtained in serious cases from the subcutaneous or intravenous infusion of normal salt solution. We are too liable, however, to forget that this might not be necessary if we persistently use the normal salt enema every day or several times a day. The great trouble in our practice is the fact that we do not anticipate danger often enough. Too many of us treat typhoid fever indifferently until the serious phases appear; then our efforts are often too late. When tympanites is pronounced it is well to add five to ten drops of turpentine, in egg emulsion, to each normal salt enema. This, with turpentine administered internally in the third decimal potency, will invariably correct this symptom.

Treatment of Special Symptoms.—When certain symptoms are extreme our persistent effort toward their removal will be demanded. *Headache* is often an early and continuous feature. If this does not respond to the use of such remedies as nux vomica, iris, belladonna, spigelia, glonoin, gelsemium, baptisia and bryonia, etc., it may be wise to resort, with due precaution, to some of the analgesics such as phenacetin and other coal tar derivatives in two or three grain doses occasionally. A favorite combination of mine is fareol 1x, iris 2x and ignatia 2x; three of these tablets administered in half hour repetitions, will often stop the headache in three or four doses. In obstinate and severe headaches we may be obliged to resort to the use of sodium bromid in ten grain doses or small doses of chloral. *Insomnia* will likewise demand some special attention, as sleep is necessary for satisfactory progress in the treatment. Usually the cool sponges will help this, but often the milder sedatives like trional, chloralamid, sulfonal, or chloretone are usually sufficient, though in exceptional instances it may be necessary to give a quarter grain of morphin or codein hypodermatically. The *delirium* may also be so serious that special medication is required. When a typical typho-mania exists the free use of alcohol by mouth and hypodermatically will often be corrective. The icebag applied constantly to the head will be the best adjuvant in such cases. The internal administration of *belladonna, hyoscyamus, stramonium, valerian* and *moschus* should not be overlooked as these will usually suffice in their various potencies. The hypodermatic use of hydrobromate of hyoscin (gr. 1/200 to 1/100) every two or three hours, is often required. The bromids may also be demanded in obstinate cases. If the indicated remedy is persistently used from the very beginning and if the adjuvant treatment, as previously suggested, is properly carried out, the serious delirium of typhoid is generally avoided.

Vomiting sometimes is a bothersome symptom, but regulation of the diet and the employment of such intercurrent remedies as *arsenicum, pulsatilla, ipecac, iris* and *cocculus* will usually remove it. In the early stages persistent vomiting may suggest the necessity of a cathartic. For mild attacks hot water with a little bicarbonate of soda is usually sufficient. *Apomorphia* in the third decimal potency is always a last resort. When vomiting takes place during the fastigium it may signify that the milk is not properly digested and this will require the addition of lime water, pepsin or further dilution. Should the stomach become excessively irritable, an absolute suspension of food may be required for twenty-four hours or longer or rectal alimentation may be demanded. *Diarrhea* is so frequently a symptom of typhoid that we should not attempt to check it unless the movements are abnormally frequent and debilitating to the patient. If the stools show undigested matter, it may be a safe deduction that an improper food is being used or the patient is overfed. Catarrhal lesions in the large bowel may be at fault, though these should be corrected by the daily enemata and the intestinal antiseptic. The indicated internal remedy should first be employed to correct this perversion. For that purpose we may use *arsenicum, cuprum ars., plumbum acet., bismuth, terebinth, mercurius, sulphur, dioscorea, cantharis, opium* or other similarly acting remedies. Opium in physiological doses may be required if no other means avails. Quarter or one-grain opium suppositories are also to be thought of in serious cases. The sulphocarbolates combined with the subgallate of bismuth is a valuable adjuvant and may be safely given three or four times daily, in doses of five grains of the former with five to ten grains of the latter. Rectal injections of astringent solutions such as tannic acid (1 to 2 drams to a quart of water) may be employed every twelve hours in extreme cases. *Constipation* will sometimes require attention though the enema and the proper diet will often correct this complication. When, however, it is obstinate, some of the salines or cascara may be employed. I do not believe in the use of calomel except possibly as an initial procedure to procure thorough intestinal evacuation. It may be indicated also when there is persistent high temperature with constipation. It is often astonishing what results may be obtained by the internal use of *sulphur, nux vomica, lycopodium* and *plumbum.* I wish again to call attention to the usefulness of such remedies as *chinoidin, euonymin, iris versicolor* and *chionanthus* in these conditions of constipation due to hepatic inaction. Elaterium (gr. 1/8) may be administered two or three times daily with good results in these cases. Elaterin (gr. 1/16 to 1/10) two or three times daily may give more prompt results.

Tympanites is a constant source of apprehension in the treatment of typhoid and sometimes it is an obstinate symptom to manage. With the proper diet, the use of an intestinal antiseptic, the regular morning enemata, this trouble is generally obviated. When it is extreme it is invariably corrected by the use of turpentine (10 drops in the egg emulsion), introduced with the enemata. In addition to this the same remedy should be administered internally in frequent doses of the third decimal preparation. Other remedies like *carbo veg.,* *lycopodium, antimonium crude.,* and *china arsenicum* will be frequently indicated. A change from a milk diet to albumin water and meat juices may be required before relief is obtained. *Hemorrhage* from the bowels will demand prompt and heroic treatment. All stimulants and food must be suspended. The ice-bag—or better, the ice coil—should be applied to the abdomen and ice should be given freely by the mouth. Opium should be given without delay to temporarily arrest the peristaltic action; this may be employed best in quarter-grain doses combined with one grain of lead acetate; it may be repeated every three or four hours until we are sure of the arrested peristalsis. The hypodermatic use of morphin (gr. 1/4), with atropin (gr. 1/150) will probably give more prompt effect in this respect. Slight hemorrhages may be corrected by turpentine given frequently in low potency. Ergotin has been almost entirely discarded in recent practice, but the aqueous solution of hamamelis in teaspoonful doses is often efficient. Adrenalin chlorid may be given in doses of 1-10 minims hypodermatically or by the mouth, but it must be used cautiously on account of its increasing the blood pressure. In serious cases of collapse as the result of hemorrhage the saline infusion must be resorted to. The internal administration of remedies according to the homeopathic theory is certainly of value in this condition. *Nitric acid* and *ipecac* in the third decimal potency, or *secale, erigeron* and *hamamelis* in the first decimal potency are of foremost importance in this respect.

Peritonitis due to perforation is a serious symptom and in the majority of cases, if given medical attention only, a fatal result will ensue. It is gratifying to know that operation if not delayed too long, will save a fair percentage of cases. As soon as the symptoms suggest this condition and particularly when there is an acute development of pain, with generalized abdominal rigidity, the surgeon should be called without delay.

Pneumonia is liable to be a complication of typhoid in many instances, and the treatment should be carried out as outlined under that subject. The same is true of *bronchitis* while *hypostatic congestion* will frequently call for such remedies as *strychnia, digitalis, atropin* and *adrenalin. Laryngitis* will demand special attention. Bedsores and thrombosis of the femoral vein are occasionally complications. The first of these is easily treated by the internal remedies and adjuvant local measures; the bedsores are always prevented by the change of the patient's position and the protection of irritated parts, while thrombosis may be relieved by the proper elevation of the limb.

The *management of the convalescence* will often require more attention and discriminating care than the periods of febrile action. It is at this time that we are liable to relax our systematic and mandatory management of the case, as the patient begs for food and more liberty. We are too frequently influenced by the solicitations of sympathetic friends. It may be stated as a safe principle of procedure that our patient should be kept in bed and on the limited diet for at least two and possibly three weeks after the temperature has remained normal. Then there must be permitted only a gradual resumption of food and a limited amount of activity. Some of the most fatal relapses have occurred at this time. Stimulation and tonics may be demanded in convalescence more than during the febrile period; therefore a light wine, whiskey or champagne may sometimes be permitted. The iron tonics are often conducive to a restoration of strength and we should never overlook the value of *arsenicum, china, nux vomica* and similar remedies at this period. As the typhoidal symptoms subside a protracted asthenia may be attended with an evening rise of temperature and a morning sub-normal fall. In such conditions anorexia is usually pronounced and the lack of nourishment is the cause of the condition. In that case we are justified in pushing our tonics to the limit and carefully permitting the patient to take more food. Post-typhoid nervous symptoms are often alarming and need special medicinal attention. The emotional and hysterical phases are often more serious than would be naturally assumed. As the patient gradually recovers from this long siege of fever it is essential that we should take into thorough consideration these nervous results of protracted infection and inanition. A post-typhoid neuritis is a common cause of anxiety, but it yields usually to continued rest, light massage and such remedies as *agaricin, zinc phos, picric acid, cimicifuga* and *spigelia.* A spinal exhaustion or neurasthenia may give rise to much physical weakness, but it is amenable to treatment as outlined under the chapter on neurasthenia.

The General Consideration of Remedies.—The medicinal treatment has already, in part, been referred to. It is important, however, to take a wider view in this respect. While the general tendency in treatment is to use little medicine, it is my belief that the homeopathic remedy has a decided sphere of usefulness in conjunction with the general treatment which has been mentioned on previous pages. No two patients suffer exactly alike nor do they respond equally to the same remedy. For this reason it will be necessary to make a careful individualization of the variations in pathological expressions and toxin effects in order to select a proper remedy. This, however, is not always possible and for that reason we must have a good understanding as to the general adaptability of remedies. If I were to mention my favorites I should speak first of *baptisia* and next of *arsenicum.* The former in its typical symptomatology conforms so closely to the picture of typhoid that we almost see the disease when we recall the delirious stupor, temperature, dry tongue and general signs of a slow intoxication. To my mind it should be used for its antiseptic effect and for that reason the tincture is the best form. When it is once indicated it should not be discarded for intercurrent remedies and it is often advisable to continue it through the febrile stage. In line with this remedy we should consider *rhus tox, echinacea, gelsemium* and possibly *arnica* and *cimicifuga.* In the study of *arsenic* we shall find that this remedy has the best effect when the temperature begins to subside. It should come in when the tide is beginning to turn toward exhaustion and circulatory enfeeblement. It is a tonic for the heart when the myocardium begins to degenerate and this is of vital importance in the treatment of functional murmurs which are liable to ensue at this time. I do not mean that it has such a stimulating action as remedies like digitalis convallaria and strychnia, but its gradual sustaining influence is better. It may not aid in the restoration of hemoglobin like ferrum, but its vasomotor superiority is evinced by its more perfect action on the arteriole structure. As a general tonic it applies with equal force to the alimentary weakness which certainly needs attention in this disease.

Ferrum phos., phosphorus, phosphoric acid and similar remedies will apply more particularly during the stage of convalescence. *Bryonia* is often serviceable in the early febrile period when the first signs of digestive disturbance are in evidence. It will be indicated by the intense thirst, dry mouth and tongue, marked restlessness and a tendency to bronchial or lung disturbances. *Arnica* is more frequently indicated in typhoid than a casual consideration would infer. The bruised sensation, the presence of boils and bedsores and the general toxic condition are all reliable symptoms indicating this remedy.

The peculiar characteristics of the fever and delirium will in some stages demand a careful consideration of *belladonna, hyoscyamus* and *stramonium.* The *belladonna* patient invariably presents the symptoms of cerebral excitement, with burning heat in the head and redness of the face. The throbbing carotids, the extreme drowsiness which may suddenly change to violent delirium, the inability to sleep or the frequent starting during sleep, the trembling of the tongue and the sore throat with bronchial irritation are other symptoms which suggest this remedy. *Hyoscyamus* will be demanded when belladonna fails; the delirium has a more furious character, involuntary twitchings and sphincter incontinence are liable to be present while the mental state may be defined as apathetic. *Stramonium* follows when these symptoms are exaggerated and the temperature is higher. I have seen this temporarily reduced by this remedy in a few hours. While the delirium may be more pronounced than in the case of either the belladonna or hyoscyamus patient, the mental feature is characterized by a more cheerful state. All of these remedies act best in the lower potencies. The *hydrobromate of hyoscin* in 1 grain doses of the third decimal, an hour apart, or in 1/100 grain doses in severe cases, will often overcome the most extreme nervous excitement and to a certain extent subdue the other serious symptoms. When there is a fixed hallucination and the patient is inclined to be loquacious, with a tendency to use violent language, no better remedy than *anacardium,* in the first to third decimal potency, can be found. In case the mental perversion takes the form of depression, *conium* may be indicated. *Agaricin* also has a decided influence over the delirium when it is not severe but constant, and is attended by a tremulous condition of the whole body. Its decided action upon the heart makes this remedy unusually efficient in the exhausted states.

Terebinth is a favorite remedy of mine, inasmuch as it corrects the tympanites and, therefore, has marked antiseptic features. It will also be indicated by the dry and red tongue, the scanty and bloody urine, and other signs which point to acute kidney involvement. *Cantharis* may supplant this when there is marked evidence of irritation in the urinary tract. The third potency of the former and the first potency of the latter are the best forms for administration. *Rhus tox.* will naturally be thought of when the patient is constantly changing his position to gain relief and it will be further indicated when the septic condition is so pronounced that the patient is affected by violent delirium or the somnolence of extreme sepsis. The third and possibly higher potencies should be employed. *Echinacea* is a similar remedy, but it may be used in more heroic doses. Ten to fifteen drops of the tincture have been given as often as every two hours with marked results. *Gambogia* has been compared to this when the gastro-intestinal disturbances are prominent, but no definite experience has been yet related regarding its usefulness in typhoid.

The *hemorrhagic condition* is generally corrected by the specific treatment already defined and the conjoint use of *nitric acid* in the third decimal potency. When terebinth has been prescribed in the earlier stages we are less liable to hemorrhage, but should this emergency arise we may need to use other remedies, among which we may favorably refer to *hamamelis, ipecac, hydrastin hydrochlorate, cinchona, ergot* and *opium* in accordance with indicated symptoms. It would be quite natural to refer to many other remedies which may, under their provings, be suggested in the various stages of the disease. These, however, will recommend themselves to the alert practician whenever they are demanded. We must keep the fact constantly in mind that as we limit the diet and give our patient scientific care we shall be able to rely less and less upon any internal medication. This, too, is an axiom which sooner or later will guide us in the treatment of all diseases.

PARATYPHOID FEVER.

This is a form of infectious fever which is clinically similar to typhoid, but it is caused by a bacillus whose characteristics are intermediate between the typhoid and the colon groups and is, therefore, called the paratyphoid or paracolon bacillus. The symptoms simulate those of typhoid, though they are more mild in character. The period of incubation is shorter and the onset is more liable to abruptness than in the case of typhoid. Diarrhea is a prominent symptom, the temperature rises rapidly and has no semblance to the typhoid curve. Mental dullness and apathy develop early and for a while are extremely intense. The spleen is somewhat enlarged, rose-colored spots may appear and many of the nervous features of typhoid may be recognized.

The duration of the fever is short and it terminates by crisis or at least a rapid lysis. The convalescence is not usually protracted and relapses are not common. Myositis and purulent arthritis are occasional complications, but intestinal ulceration is not a part of the pathology. The Widal reaction, of course, can not be found but the serum reacts upon fresh cultures of the paracolon bacillus. The management of the case may be conducted according to the general suggestions given under typhoid fever.

MOUNTAIN FEVER.

Mountain fever, as its name infers, is found in the mountainous region of the West. It is apparently a variety of typhoid which is modified by the influences of a high altitude. Some regard it as purely indigenous to these regions and without any definite pathology resembling typhoid. The lesion of typhoid has, however—in a very few cases—been found in the ileum. The symptoms bear a slight relation to those of typhoid, but are in no sense so pronounced. Epistaxis frequently ushers in the fever, and headache, exhaustion and malaise prevail during the early stages, the pulse is rapid, but the temperature at no time reaches a very high degree. The treatment will be similar to that of typhoid, but the return to a normal altitude will generally be all that is required to accomplish a cure.

TYPHUS FEVER.

This disease has been variously defined as ship fever, camp fever, jail fever and exanthematic typhus. It is an acute infectious disease of unknown origin, highly contagious and characterized by its sudden onset with rigors and high fever, the early prominence of nervous symptoms, the maculo-petechial eruption appearing between the third and fifth days and the termination by sudden crisis about the end of the second week.

History.—Of all the great epidemics which have ravished the European countries down to the middle of the nineteenth century, typhus has been the most prolific scourge. It may be traced back beyond the Christian era, as the plague of Athens (B. C. 470 to 400) which was described by Thucydides, was no doubt due to this infection, while the Scriptural reference to such a pestilence gives frequent evidence of its occurrence as a plague. Russia, Ireland and parts of England suffered frequently with its devastations, and in 1812 it was first discovered in the New England States of our country and spread with terrible results over the entire country. In 1836 it reappeared with great virulence, but its spread was not extensive and this country has been afflicted only with isolated cases since that time. The same is true of European countries and we owe this fortunate circumstance to the improvement in sanitary conditions and the triumphs of modern medicine. Few modern students of medicine have had the privilege of studying this disease and it is probable that many of the supposed epidemics which develop in small groups are nothing but aggravated types of typhoid.

Etiology.—The *direct cause* has not yet been found, though it is probably induced by a specific poison elaborated in the body of the patient. While bacteriological investigation has produced no indisputable evidence there can be no doubt that it is established by the agency of a pathogenic micro-organism. When it does exist it may always be traced to a case of typhus and does not arise spontaneously. The same uncertainty exists relative to the organs and tissues which are particularly affected, and as yet we do not understand how the poison leaves the body affected or enters that of a healthy person. The *dissemination* is supposed to come directly from the expired air and the skin exhalations, the dust acting as a medium of conveyance into the respiratory tract of anyone who has been exposed. The contagion is so distinct that direct contact with the patient is liable to develop it; it may likewise be transferred by means of clothing, furniture or attendants who have cared for a patient, and this explains how the poison may remain in any surrounding for a long time and thus affect one who is susceptible. For this reason complete isolation must be established in every epidemic.

Predisposing causes have a pronounced influence in developing the disease and chief among these may be mentioned the filth and unsanitary surroundings which are found in the crowded and poor quarters of a city. Famine has also been a decided factor in its serious epidemic development, and in some respects it is encouraged by intemperance, overwork and nervous influences. Age and sex seem to have no relation to its occurrence, though the young and middle aged have furnished the greater proportion of cases. The worst epidemics have taken place during the winter months, though there is no special predilection toward any particular season.

Pathology.—There is no distinct morbid anatomy. Rigor mortis may be delayed, the petechial eruption may remain for some time after death and ecchymotic spots or in some instances gangrenous bedsores may be observed. The anatomic changes are usually those which result from the intense fever and secondary infections. The pericardial and gastro-intestinal mucosa are often the seat of minute hemorrhages while hemorrhagic extravasations in the muscles and especially in the myocardium may give rise to marked granular degeneration. The liver suffers a moderate enlargement while the same is true of the spleen and kidneys. The lymph follicles are occasionally enlarged, but ulceration does not follow. The blood is very dark in color, its fluidity is increased and its coagulability is decreased. Hypostatic congestion of the lungs is a frequent sequence and bronchial catarrh is commonly noticed.

Symptoms.—The *incubation* covers a period of about twelve days, but it may be confined to a shorter time. A slight feeling of discomfort may constitute the prodrome, but the invasion is generally suddenly announced by a chill or rigors and the immediate appearance of a high fever which attains its maximum (105°-106° F.) at least by the second day. In this we shall find no sign of the temperature curve which characterizes typhoid. The chills may recur and be attended by marked pain in the head and back. The prostration is extreme and the patient takes to his bed early. The pulse is full and rapid in proportion to the temperature. The tongue is heavily coated, the face is flushed, the conjunctiva is congested, vomiting frequently occurs and the facial expression is dull and stupid. Bronchial catarrh is common and the urine is often scanty with a high specific gravity and a trace of albumin.

The nervous symptoms are recognized at the outset and are often severe. Mild or very active delirium may appear at the onset of the fever and gradually shade off into a stupor or an actual coma, either of which gives a facial expression which is distressing to look at.

The *eruption* is discovered between the second and the fifth day and with its appearance there is no noticeable decline in the temperature. The rash will be found first upon the abdomen and upper part of the chest and then upon the extremities and face; its development is rapid and usually complete by the third day. The eruption has two characteristic features. First, there are distinct papular rose-spots which change to petechiae and later assume a very dark hue on account of the hemorrhagic extravasations. These have a more extensive distribution over the body than the typhoid rose-spots; they are less likely to disappear on pressure and the color is much darker. Besides the petechial eruption there will be a decided dark mottling of the skin which, with the lighter and darker shades in alternation, will give a peculiar mulberry appearance. Inasmuch as the rash is hemorrhagic it does not disappear after death. In the milder grades of typhus the rose-spots may not become petechial and, therefore, the disease is often misunderstood for typhoid.

The *second week* usually shows a marked aggravation of all the symptoms. The fever continues high, the prostration increases, the tongue becomes dry, sordes form about the teeth and the delirium becomes more intense. The stupor deepens, the face is flushed, the conjunctivae are injected and the pupils are contracted. Subsultus, nystagmus, picking at the bedclothes, accelerated respiration and feeble cardiac action are soon noticed at this time. Coma-vigil—a condition defined by the half-opened eyes and complete unconsciousness—and the peculiar odor of typhus are also recognized during the extreme stages. This condition may continue until death takes place as the result of adynamia. In favorable cases about the end of the second week a crisis occurs suddenly, the temperature declines rapidly and the patient awakes, after a refreshing sleep, apparently refreshed and with the mind clear. Convalescence then ensues slowly and relapses are uncommon.

The *character of the fever* has been defined by its rapid rise on the first day. While this is true it does not always reach its maximum before the fifth or the sixth day. In extreme cases the temperature may be as high as 107° F. After the maximum is obtained, slight morning remissions take place until the twelfth or fourteenth day, when the crisis suddenly appears. When the temperature reaches 108° or 109° F., we may expect a fatal termination. *Cardiac weakness* must be expected in connection with the febrile excitement and this will usually be distinguished by the feeble first sound and the recognition of an apex systolic murmur which is not organic. Variations in the character of the disease often occur. There is a malignant form known as *typhus siderans* which proves fatal in two or three days, and on the other hand every epidemic is liable to present a great many mild cases which terminate favorably in about ten days.

Complications are not numerous in this disease, but there are some which need special attention. *Bronchitis,* hypostatic congestion and particularly broncho-pneumonia are liable to cause serious apprehension during a run of typhus. *Pulmonary gangrene* may be a sequela and this extending by contiguity may induce a fatal empyema. In like manner a sero-fibrinous pleurisy or a lobar pneumonia may be a secondary development. Gangrene involving the fingers and toes may also occur. The *digestive tract* suffers also some associate disturbance. Hematemesis, cancrum oris and parotitis are occasionally observed, while the septic invasion of the intestine may give rise to serious conditions of enteritis. The *nervous phenomena* are particularly marked by a delirium which often reaches the comatose or the maniacal stage, but in addition we may occasionally meet with meningitis and neuritis followed by paralysis. *Hemorrhagic nephritis* though of rare occurrence, is a complication which should always be feared.

Diagnosis.—To determine a case of typhus fever will not be an easy matter for a practician who has had no experience with the disease. As that applies to the majority of us in present day practice, on account of the rarity of the disease, the diagnosis is doubly doubtful. The knowledge of an epidemic will first call our attention to the true nature of the disease and then we shall be able to decide only by the characteristic symptoms already outlined. The most cardinal of these are the character of the eruption, the abruptness of the invasion, the rigor followed suddenly by a high fever, the early appearance of marked nervous symptoms and the sudden termination by crisis. *Typhoid fever* will be distinguished by its gradual onset without any marked chill, its milder and later developed nervous symptoms, the less abundant eruption, its later appearance and the absence of petechial signs, the positive Widal reaction and the characteristic fever. *Cerebro-spinal meningitis* will be recognized by a more intense headache, retraction of the head, hyperesthesia, intolerance of noise, photophobia, strabismus, tendency to convulsions and paralysis, the non-petechial eruption and the absence of coma-vigil. Kernig's sign and the microscopic findings, following a lumbar puncture, will settle the doubt. *Malignant variola* may simulate this for a short time, but the greater extent of the hemorrhages and the bleeding from the mucous membranes will clear the doubt. *Malignant measles* has a close resemblance, but the eruption is brighter, often crescentic or irregular in arrangement and appears first upon the face. *Uremia* has a previous history of renal incompetency, there is no eruption, while edema and other urinary phenomena are present. *Bubonic plague* resembles typhus in its extreme fatality and the suddenness of its onset and fever; the eruption, however, differs in that it quickly becomes carbuncular.

Prognosis.—The mortality of typhus is usually high, but there are some epidemics in which the death rate is not higher than twelve to twenty per cent. Those beyond middle life are more liable to death and in a majority of cases among children a favorable result may be looked for. When death occurs during the early stage it is probably due to the toxemia but when it occurs later it is more liable to be the result of some complication like pneumonia. A fatty degeneration of the heart predisposes to a sudden death. It is impossible to say what the mortality would be, in typhus epidemics, under our present medical technique and nursing.

Treatment.—The most important part of the treatment is a *proper isolation* for the sake of others. For this reason camps are supposed to be a necessary means for the separation and quarantine care of such serious epidemics as typhus fever. It is fortunate, however, that under present sanitary conditions, these epidemics are becoming very rare indeed. Much of the technique which applies in typhoid may be used with advantage in these cases and, therefore, hydrotherapy is a valuable expedient, but it is improbable that dietetic limitations should be carried to such an extreme as in typhoid. *Stimulation* is, without doubt, a very important feature of the treatment, as it is necessary from the very beginning to resist the asthenia. Alcohol is probably our best agent for this purpose, though quinin, strychnia and digitalis may be considered in that respect. Antipyretics are dangerous remedies inasmuch as the cardiac depression caused by these may be hard to overcome. For complications remedies may be used in accordance with symptoms. Such remedies as baptisia, echinacea, arnica, arsenicum, lachesis, crotalus and naja should be studied.

RELAPSING FEVER.

Various terms have been used in the past to define this fever which has been referred to as famine fever, recurrent typhoid, bilious typhoid and spirillum fever. It is an acute infectious disease characterized by febrile periods lasting only a few days and separated by periods of total remission for about the same length of time. It is caused by the inoculation and multiplication of the spirocheta of Obermeier.

History.—The history of this disease dates back as far as that of typhoid and typhus. It prevailed extensively in Europe, particularly in Ireland, and it is more common in India than any other part of the globe. It was observed in Philadelphia, Pa., in 1844 and twenty-five years later a serious epidemic was experienced in New York. Since this time we have no history of its existence in this country. The predisposing causes are similar to those which favor the development of typhoid and typhus, as the lack of hygienic surroundings and imperfect food favor its promotion and rapid spread. It is contagious as well as infectious but sex, age and seasons have no influence in its propagation.

Etiology.—The specific cause was discovered by Obermeier, who found the micro-organism which has been named the "spirillum Obermeieri." "This specific agent or spirocheta, is a delicate filamentous organism of spiral form and much elongated, its length equalling four to six times the diameter of a red blood corpuscle. Under the microscope it will exhibit active spiral motion but it is found in the blood only during the febrile paroxysms." Inoculation may be accomplished by the spirilla in this period of their activity but they lose their inoculating power during the apyrexial period. It is presumed that the organism is given off in the breath or from the skin.

Pathology.—No characteristic anatomical lesions have as yet been discovered. When death takes place during a febrile paroxysm the spleen is found to be large and soft, while the liver, heart and kidneys present a cloudy swelling and occasionally a bloody extravasation. The bone marrow may be in a state of hyperplasia and ecchymoses are sometimes discovered.

Symptoms.—The *period of incubation* varies in its duration and is characterized by a marked sense of malaise. It is frequently short or in some instances it may not be recognized. The *invasion* is usually abrupt and is characterized by sudden chill, fever and intense pain in the back and limbs. Dizziness, severe headache, vertigo and pronounced prostration soon intervene. The temperature rises rapidly to 104° or 106° F. and the profound depression causes the patient to take to his bed. The pulse rises rapidly, nausea and vomiting are common symptoms and convulsions are liable to appear in young subjects. The skin is dry and dirty yellow in color, the face is flushed, perspiration is profuse while sudamina are often present. Herpes is sometimes detected and the same is true of petechiæ though there is no characteristic eruption. Jaundice is a rare symptom, abdominal tenderness is sometimes observed and physical examination may reveal slight hepatic and splenic enlargement. Cardiac weakness may often be detected by the irregular and intermitting pulse and hemic murmurs are audible in some instances. Nervous symptoms are not pronounced though delirium of a mild grade is occasionally present. During the paroxysm the spirilla will always be found in the blood.

The *fever* always terminates suddenly by crisis. This occurs about the fifth or seventh day and if often announced by a marked rise of temperature. It is evidenced by a profuse sweating which is followed by a rapid fall of temperature to normal or subnormal. In place of the copious perspiration the crisis may be ushered in by diarrhea, epistaxis, intestinal hemorrhage, or metrorrhagia. The afebrile period appears in a few hours or at least in a day and a speedy convalescence seems to be in evidence.

The *relapse* occurs in about a week after the crisis or on the fourteenth day after the primary chill. This is characterized by a repetition of the febrile paroxysm and its subsequent crisis. This seizure may, in turn, be followed by one or more relapses with similar terminations but usually the paroxysms are limited to two or three in number. In general each succeeding attack is shorter than the previous one and occasionally the disease terminates with the first crisis. Convalescence is also of a rapid character and the duration of the disease as a whole is rarely beyond three weeks. One attack, however, does not furnish immunity from another.

Among the *complications* which may follow are bronchitis, broncho-pneumonia, pneumonia, nephritis and hematuria. Epistaxis is sometimes a persistent sequela and other complications of a suppurative character may be enumerated. As a natural result of the high temperature, sudden crisis and toxemia the heart may manifest great debility.

Diagnosis.—The peculiar development of the fever with its early and sudden crisis followed by a stage of afebrile convalescence and then a relapse will distinguish this fever from both typhoid and typhus. The positive confirmation of relapsing fever is made by the discovery of the spirilla. The other symptoms previously enumerated should help to confirm the diagnosis. When *typhus simulates* it will be differentiated by the injected conjunctivae, the contracted pupils, the stupid condition, the characteristic maculo-petechial eruption and the marked delirium. The period of febrile action is also longer and a relapse is uncommon. *Yellow fever* has some resemblance to this but the stage of remission is shorter, poorly defined and characterized by the black vomit and intense jaundice. *Malarial fever* may have some resemblance to the relapsing form but the microscopic revelation will correct this confusion.

Prognosis.—The prognosis in relapsing fever is not entirely unfavorable though much depends upon the severity of the epidemic and the previous debility of the patient. Complications like pneumonia and nephritis of course may contribute toward its unfavorable termination and at best we should regard it as a serious disease. It is possible for a patient to die during either the paroxysm, the crisis or the relapse, though this fatality is not usually to be expected. Severe hemorrhage, uremia, extreme jaundice, excessive vomiting, persistent diarrhea, and syncope must always be regarded as alarming symptoms.

Treatment.—The principles which pertain in the treatment of all infectious diseases certainly apply in the management of this disease. Febrile remedies will be of use in accordance with their symptoms. Therefore, we may expect to use such remedies as *aconite, bryonia, belladonna, baptisia, veratrum viride, rhus tox, cimicifuga, echinacea,* and *eupatorium perfoliatum.* Stimulants may be employed when the depression is pronounced the same as in typhoid fever and we may also expect to relieve the high temperature by means of baths and cold sponges. Inasmuch as delirium is rarely present we shall not have so much use for the ice bag and other adjuvants which hyperpyrexia often demands. As the lesion is not found in the intestine the requirement of normal salt enemeta will not be so mandatory though they are certainly useful for the sake of bowel movements and intestinal cleanliness. *Dietetic care,* though not so important a consideration as in typhoid, is still of much significance as a liquid and limited diet will be required in the febrile stage. Care must also be exacted during the afebrile period lest an attempt to overnourish favors an early relapse. The fact that the disease is almost extinct in this part of the country accounts for our limited knowledge regarding the requirements of treatment.

MALTA FEVER.

The above name has been given to this fever because it has appeared in the endemic form at Malta and other Mediterranean ports. It has also been known as rock fever and undulant fever. It is caused by the micrococcus melitensis and is clinically characterized by an irregular fever with copious sweats, rheumatoid pains and frequent relapses. The disease has also had some epidemic sway in various parts of Europe as well as the United States, but its environment is rarely confined to the locality which has contributed the name. No pathologic lesions have been established as typical features of the disease though it has been regarded as a pseudo-typhoid or malarial fever inasmuch as splenic enlargement has been so frequently found. Positive evidence of any other similar pathological relation has not been demonstrated. Young people and healthy adults are principally affected and anti-hygienic surroundings naturally favor its development.

Symptoms.—A short *period of incubation* prevails before the initial symptoms appear. The *onset* is gradual and the patient at first complains of headache, anorexia, sleeplessness, thirst and slight chilliness much the same as in the early stages of typhoid. *Epistaxis* is an occasional symptom, the spleen is often enlarged and tender to pressure and the bowels are constipated while the stools are frequently streaked with blood. The *fever* is not very high at first and is distinctively of the remittent or undulating type.

These symptoms last from three to four weeks when the *first remission* sets in; this continues only for a few days when the *first relapse* appears and this is characterized by rigors, high fever and sometimes diarrhea. This relapse lasts usually from five to six weeks and is followed by *another remission* of ten days or two weeks. Then the *second relapse* follows with symptoms similar to the first relapse. In addition, however, symptoms of debility, night sweats and pain in the larger joints are now experienced. Then comes the *third remission* which may last a month or longer. The *third relapse* then sets in but it is of shorter duration, the temperature has an afternoon aggravation followed by night sweats and a morning normal temperature. The tongue is heavily coated, the rheumatic pains are extreme and the joints are swollen and extremely painful. The fibrous tissues are often involved and this will be particularly noticed about the ankles and the tendo Achilles.

Diagnosis and Treatment.—By the means of pure cultures of the special organism a typical agglutination will take place with the blood of the patients affected by this fever. In this way a proper differentiation will be made between this and typhoid. Otherwise there would be much confusion inasmuch as the disease is of such rarity. The prognosis is usually favorable and the treatment, with the exception of combating the exhaustive effects, will be purely symptomatic.

MALARIAL FEVER.

Malarial fever has been defined by many other terms such as "chills and fever," "fever and ague," "swamp fever," etc., all of which have reference to the Italian origin of the word meaning bad air. A more recent knowledge of the disease has confirmed the definition of an infectious and non-contagious disease which is caused by the hematozoa of Laveran. It is characterized by paroxysms of intermittent fever of the quotidian, tertian or quartan type, a continued fever with marked remissions, a pernicious or rapidly fatal form, and a chronic cachexia with anemia and enlarged spleen. Six varieties are presented for study: (1) Intermittent fever; (2) Pernicious intermittent; (3) Remittent fever; (4) Malarial cachexia; (5) Masked intermittents; and (6)Malarial hematuria. It is a disease of ancient history and has been most prevalent in localities adjacent to swamps and marshes, the home of the mosquito. It is fortunately disappearing as the advent of civilization is reclaiming the marshy districts through the process of drainage and sanitation. Our modern knowledge of its cause has helped us to thwart its growth and our more perfect therapeutic understanding has taught us how to limit the severity of its attack. That it is a source of alarm so long as the predisposing conditions exist can not be denied and civic energy and professional attainment must still work hand in hand to overcome its pestilential occurrence.

Etiology.—Our knowledge of the *bacterial origin* of malarial fever was first established in 1880 by Laveran, who discovered the malarial parasite in the blood of persons suffering from the disease. A few years later the developmental cycle or sporulation in man was confirmed by Golgi. He observed that certain parasites developed into peculiar crescentic bodies while other rounded bodies displayed an abnormal agitation soon after being withdrawn from the body and discharged several flagella. The significance of these crescentic and flagellated bodies was determined in 1897 by MacCallum, who learned that some of these bodies were hyaline and others granular, that the hyaline developed the flagella and when a flagellum separated from the mother cell it entered a granular body and thus established the process of fertilization. Its fertilization and development in an intermediate host has been found to take place in the mosquito and for this discovery we are indebted to the persevering efforts of Surgeon-Major Ross, of Liverpool, England. "To determine this he fed the mosquitoes bred from the larvæ on the blood of patients containing crescents and he found that peculiar spheroidal cells developed on the walls of their stomachs and this convinced him that these cells constituted the mosquito stage of the parasite (zygote). The zygote grows rapidly, protruding into the insect's body-cavity. Later the cell substance divides into ten or twelve 'meres' and in one to three weeks the zygote matures when each mere contains a number of delicate threadlike blasts. The capsule of the zygote ruptures and sets the threadlike blasts free within the insect's body when they are carried by the blood current to all the tissues, but particularly into the insect's salivary glands. As the duct of the salivary glands of the mosquito enters the proboscis the secretion of this gland is introduced into the wound caused by the insect's bite. In the human body the blasts return to the ameba with which the life history of this parasite began." The malarial parasite of Laveran belongs to a subclass of protozoa known as hematozoa; and the special varieties in their action upon the blood of human beings are described as follows by Anders:

"The ameba causing tertian intermittent fever begins its evolution in the red blood corpuscle in the small hyaline form. Gradually fine, mobile granules in the shape of pigment develop within it so that when the ameba matures it attains the size of the normal red corpuscle, and assumes a spherical form. It then sporulates into fifteen or twenty segments and as the parasites are often numerous the sporulation of an entire generation may take place in a few hours. As the parasite develops the red corpuscle is enlarged and decolorized. The *malarial paroxysm* is due to the process of sporulation and the symptoms are probably the result of a toxin development. As the tertian parasite runs its cycle in about forty-eight hours the paroxysm following the sporulation would occur every second day when the infection is by a single generation. When there is an infection by two groups of parasites on successive days a daily malarial paroxysm may follow (quotidian intermittent). In rare instances there may be a multiple infection."

"The ameba causing quartan fever is made up of coarser, darker and less mobile pigment granules. It does not attain the size of the red corpuscles and when sporulation takes place the segments, five to ten in number, encircle the pigment mass. The red blood corpuscle which is affected, instead of becoming larger and paler, contracts about the parasite and assumes a

deeper or greenish hue. Sporulation takes place about seventy-two hours after the red corpuscle is invaded. Thus if only one group of parasites exist the paroxysms—quartan intermittent—will occur every fourth day. Double quartan infection will result in paroxysms on two successive days with an intermission of one day. Infection by three groups of parasites will create daily paroxysms—the quotidian intermittent. Infection by more than three groups is rare."

"*The ameba causing estivo-autumnal fevers* is much smaller than the other varieties, often being less than one-half the size of a red blood corpuscle. The pigment is scanty and in the earlier stages of its development only small hyaline bodies, with one or two pigment granules, are found in the peripheral circulation. In the later stages the parasite may be found in the blood of certain internal organs such as the spleen. The corpuscles containing the parasites are liable to be shrunken, crenated and brassy-colored. After the process has existed for a week or more, larger, refractive, crescentic, ovoid and round bodies, with central clumps of coarse pigment granules begin to appear and these are characteristic of estivo-autumnal fever. Two varieties—the tertian and quotidian—are found in this form of fever. The parasites of the tertian variety are the larger of the two and assume a signet ring form during the hyaline stage; the ameboid movement is sluggish and one organism is usually found in a blood cell. Segmentation takes place outside of the corpuscle and the crescents are large, slender and deeply pigmented. The quotidian parasite is smaller, at times mobile, and more than one parasite occupies a single red cell which is generally wrinkled. The pigment is motionless and resembles short rods. The crescents are small and often present a double outline. Segmentation takes place within the corpuscle."

The *predisposing causes* of malarial fever need some consideration. The *soil* is naturally concerned in its development inasmuch as we nearly always find it prevailing in marshy localities, hence it is so common in the tropical countries. The *climate* has also much to do with its occurrence as the warmer localities are predisposed to the long life of the malarial parasite. In the temperate zone the majority of cases are liable to exist during the autumn, while in the tropics it prevails both during the spring and autumn. Race and sex need slight consideration for we shall find that it shows no particular favoritism in this respect. Immunity exists in many instances and a primary attack, thoroughly treated by quinin, may be regarded to a certain extent as a preventive against a second infection.

Pathology.—The blood is affected directly by the malarial parasite, the red blood corpuscle being rapidly destroyed. Pigment accumulates, not alone in the blood but also in the spleen and liver, as the result of the conversion of hemoglobin into melanin. The generation of a toxin accounts for the febrile action. The spleen is filled with blood and is decidedly smaller. This is more marked during the paroxysm but the enlargement may remain for some time after the disease is cured, giving rise to the condition known as "ague-cake." In the acute swelling hemorrhagic infarcts may take place and in rare instances the spleen has ruptured. The liver is subject to slight enlargement and the heart chambers may be acutely dilated. Other phases of the pathology will be taken up under the descriptions of the different varieties of malaria.

Intermittent Fever.—*Symptoms.* The paroxysms of this form of malaria are defined by three stages, the chill, fever and sweat, though slight or exaggerated prodromal symptoms may precede for a day or two. The *chill* is characteristic; it usually begins gradually with a chilly sensation which grows in intensity until the patient shakes violently. The lips are blue, the face is pale and pinched, the surface feels cold and malaise is pronounced. Headache, nausea and vomiting may be experienced. The occurrence of the chill is most frequent during the morning hours and it often lasts for one or two hours. The pulse is small, hard and frequent. The *fever* follows the chill and the hot stage is as pronounced as the chill. The face is flushed, the skin is intensely hot and dry, the tongue is coated, the breath is foul and the mouth is very dry. The temperature begins to rise and may reach 104° to 106° F., though in many instances it will show variations in its range. The pulse is full and bounding unless there is some cardiac weakness. The duration of this stage is from half an hour to four or six hours. The *sweating* stage starts with beads of perspiration on the forehead and face and gradually extends over the entire body. All the symptoms are now relieved, the temperature disappearing by crisis though the decline is not so rapid as the onset. Occasionally the fever drops by degrees until the subnormal point is reached. The duration of this stage is from eight to twelve hours. The paroxysms recur with unfailing regularity unless they are overcome by treatment. The usual intermission lasts twenty-four hours (quartan intermittent). These characteristics of the paroxysms may vary in accordance with the sporulation of the different groups of parasites, two paroxysms occasionally taking place in the same day. Sometimes they occur a little earlier each day when the term "anticipating intermittent" is applied, or again they may recur an hour or two later each day in which case the term "retarding intermittent" is employed. Mild forms of malaria frequently exist and these will be defined by the irregularity or absence of some of the characteristic stages. Most of these conditions are called "dumb ague."

Other characteristic symptoms are found in the *splenic enlargement* which is usually detected soon after the first paroxysm and gradually increases with the subsequent seizures. The swelling will be readily detected by palpation, and tenderness will be found upon deep pressure. *Hepatic enlargement* is not so pronounced and depends much upon the previous condition of the liver. The malarial complexion, due to the deposit of pigment, will be characterized by the yellowish-brown discoloration. *Gastro-intestinal* disturbances are occasionally experienced, diarrhea being most prominent; *paroxysmal vomiting*, attended by catarrhal jaundice, is a feature of the severe forms of intermittent. *Herpes* and other skin eruptions are often present. The *urine* contains a small amount of albumin and there is usually a temporary increase in the amount of urea eliminated two to six hours before the chill. The *heart* is subject to acute dilatation and functional murmurs may be heard during the paroxysm. The *blood change* will be characterized by the rapid diminution of both red and white corpuscles during the severe paroxysms. In children the rigor may be displaced by a pronounced convulsion and acute nephritis is a more common sequela.

Pernicious Malarial Intermittent.—This form of malarial fever is characterized by severe paroxysms but fortunately it is of rare occurrence in the present generation. It is occasionally found in the malarial districts of southern countries and, therefore, it may be found in Italy, the lower Danube, in the southern States of our country and in parts of tropical Africa. Two particular types are recognized, the comatose and the algid.

The *comatose form* may or may not have the initial chill but when it does occur it is extremely severe. The onset is sudden and marked, characterized by acute delirium or coma, due to the lodgment of the parasites in the brain in large numbers, where they form thrombi and generate toxins. The patient may perish in this state under the influence of the violent nervous symptoms and the toxemia, or again consciousness may return only to be followed in a short time by a fatal relapse. The *algid form,* often termed congestive chills, is characterized by violent gastro-intestinal irritation in which extreme nausea, vomiting, and purging are followed by collapse, intense prostration and coldness of the surfaces. In this respect it closely simulates the algid stage of true cholera. The internal temperature is very high while the surface temperature is low. Jaundice is frequently a marked symptom. The lesion is localized in the gastro-intestinal mucosa where the parasites are found in great numbers. This form is often confused with yellow fever while the comatose variety is likely to be confounded with certain phases of typhoid. In accordance with their prevailing characteristics other forms, as for example the hematuric, are also found.

The *pathology* of the pernicious malarial intermittent fever is to be studied as an infection and as a re-infection. When it exists as the result of a fresh infection the blood will be hydremic, while the corpuscles will be in a state of disintegration. The spleen is soft, swollen, turbid and unnaturally pigmented. Pigment granules and red corpuscles containing parasites and phagocytes will be found in the pulp about the arterioles. The liver is similarly affected, phagocytes and parasites containing pigment being found in the red corpuscles of its minute capillaries. The kidney will show a similar pigmentation in the neighborhood of its blood-supply and necrotic areas may also be detected microscopically. The brain may have a characteristic color or a lighter hue as it may be severely congested or often anemic; the capillaries and perivascular spaces are often infiltrated with phagocytes and disintegrated blood discs which contain parasites; under this condition hemorrhages, which constitute focal lesions, may be established. In case of re-infection the blood is often extremely hydremic while the spleen, liver and kidneys are indurated and present a decided pigmentation to the eye.

Remittent or Continued Fever.—(*Estivo-Autumnal Fever.*—This type of fever is found most frequently in the southern States and in tropical countries. It prevails in the summer and fall and is often called "bilious remittent fever" on account of the severity of the gastro-intestinal features. It is due to the estivo-autumnal parasites in the blood of the patient.

The *symptoms* are often irregular and the prodromal features are characterized by headache, loss of appetite and epigastric discomfort for several days. The paroxysms occur daily or twice a day resembling the quotidian and tertian intermittent though their duration is longer. As the febrile attacks may often be anticipating and inasmuch as the remissions have a tendency to become shorter and shorter the fever often assumes the continued form. The average case is ushered in by a chill and the fever rises rapidly, reaching its maximum (104° to 105° F.) in ten or twelve hours. The pulse is full and rapid, nausea and vomiting are distressing, epigastric tenderness is marked, the spleen is enlarged, delirium is often present and bronchitis is not uncommon. About midnight there is a marked remission in the temperature and all the other symptoms, sweating succeeding. By morning the temperature nearly reaches the normal point but an exacerbation of the paroxysms then intervenes with a repetition of the previous day's occurrence only the temperature may go higher. These phenomena recur regularly until the fever has run its course, in about two to four weeks in the average case. During the period of high febrile activity serious nervous symptoms may appear, the urine is diminished and acute nephritis is often a complication. In the severe type of this fever the paroxysms increase in their duration, thus obliterating the regular remissions and simulate the continued fever. The fever may then be prolonged for several weeks and assume more of the type of a typhoid or a pernicious intermittent. A fatal termination is liable to occur at this time though a mild form may yield to the proper treatment.

The *pathology* is characterized by abnormal pigmentary deposits in the spleen, liver, brain and kidneys, depending of course upon the duration of the infection. The spleen in mild cases is swollen and soft but in protracted cases it attains a larger size and is inclined to be hard. The pigment is most abundant in the splenic pulp about the splenic veins. The liver is also liable to assume an hypertrophic hardness when the disease is of long standing, the pigment being formed in granular masses giving the bronzed appearance. The brain is similarly affected, the arterioles being crowded with phagocytes and disintegrated blood cells containing parasites. The kidneys are involved in the same manner with evidence of acute degeneration in the cortical structure. The lymphatic glands, the skin and other tissues are deeply pigmented while the blood is hydremic and filled with degenerated blood-discs.

Malarial Cachexia.—This is a chronic condition following the repeated attacks of one of the acute forms, or it may rarely develop in malarial districts independent of the acute attacks. The *symptoms* are not clearly defined and the febrile action is rather irregular, rarely attaining a high degree. The skin has a dirty yellow color, the spleen is decidedly enlarged and indurated, the same condition applying to the liver in a limited degree. Anemia is the characteristic symptom, the red corpuscles suffering a marked reduction. The *general symptoms* will include debility, dyspnea, edema, anasarca and frequent sweatings. The nervous symptoms are often extreme, tremors, neuralgias, palsies, insomnia, and neuritis often prevailing. Gastro-intestinal disturbances, bronchitis and slight arthritic attacks are often noticed. Hemorrhages from the mucous membranes and into the retina are common while such chronic conditions as dysentery, fatty degeneration of the heart and nephritis may be dangerous sequelæ. The majority of these cases will respond to persistent treatment.

Masked Intermittent.—This is a form which resembles malarial cachexia differing only in the fact that it has no fever. The paroxysms are characterized by the periodic occurrence of pain which assumes the form of neuralgia. This may be experienced in the supraorbital, intercostal, sciatic and other nerves. The seizures are most common during the morning hours, there being a tendency to relief late in the afternoon. Convulsions or paralysis may supplant the fever in some instances and the same is true of paresthesia and anesthesia. In other cases instead of the fever, hemorrhages, edema, diarrhea, dysentery or dyspeptic crises may characterize the paroxysms. This form of malaria is difficult to diagnose and will require not alone a blood analysis but the confirmation by anti-malarial treatment as well.

Malarial Hematuria and Hemoglobinuria.—Hemorrhage is often a decided symptom in the course of pernicious malaria. Blood and albumin are frequently found in the scanty urine during the febrile stage. These may be accompanied by epithelial and granular casts or other nephritic signs. Hemorrhages from other parts of the body—the nose, mouth, rectum or vagina—may be present also while ecchymotic spots as well as an intensification of the malarial complexion may be presented. Prostration is usually extreme, nervous symptoms are pronounced, uremic toxemia may appear in case of urinary suppression and a patient may die suddenly from heart failure or as a result of the hemorrhage.

Hemoglobinuric fever has been discovered among soldiers in malarial districts. In these cases the hemoglobin suffers a marked reduction, the majority of the red corpuscles revealing the presence of parasites and with this severe hematuria may be associated. The usual symptoms are characterized by a mild chill, a sub-febrile temperature, hematuria and frequently hemoglobinuria. The paroxysms may occur each day, oftener, or at longer intervals. The hemoglobinuria is usually a continuous feature though it is aggravated at periods. The semicomatose condition is a prominent symptom and in case of urinary suppression—which often comes on suddenly—uremia may follow. The presence of malarial parasites in the blood and the hemoglobin in the urine constitute the important facts in diagnosis.

The *complications* of malaria are not many. Nephritis and rheumatism seem to have a peculiar relation to this disease and typhoid is often a complication though it has no particular relation to malaria. Pneumonia is of frequent occurrence but this too apparently develops independently. Enteritis is probably a more common sequela while jaundice and dysentery are frequently associate conditions. Among the rarer complications the nervous sequelæ should be mentioned. Paraplegia often follows from a peripheral neuritis or even a cord change which may develop at this time. Hemiplegia may occur in the pernicious comatose form or during the height of a severe paroxysm. Spiller has described an acute ataxia and a disseminated sclerosis and Osler gives evidence of multiple gangrene occurring in conjunction or as a sequence of malaria.

Diagnosis.—The diagnosis in a case of malaria is not so difficult under the modern diagnostic methods. The characteristic symptoms which define the paroxysms should call attention to the probability of this disease. This should be doubly true when the patient resides in a malarial district. The most confirming evidence, however, is furnished by the microscope for by this it is possible to detect the parasite in all its stages in the blood of the patient. Facilities for this observation are fortunately now within the reach of the majority of practicians. When these facilities are not at hand it is often necessary to make differentiations by the study of symptoms and physical findings. *Pyemia* may be recognized by the less regular recurrence of the chills and the more profound prostration. In this we shall find a leukocytosis which is absent in malaria. *Acute tuberculosis* manifests its pyrexia in the afternoon and chills are not a prodromal feature of its onset. The general symptoms, the history, the chest findings and the leukocytosis will also help the differentiation while the administration of quinin gives no improvement. *Ulcerative endocarditis* may present an intermittent pyrexia but the disease is more severe, leukocytosis is present, quinin gives no relief and the attending cardiac symptoms give more positive diagnostic suggestions. *Typhoid fever* will often simulate the remittent malarial fever but its more gradual onset, the slight remissions, the characteristic eruption, the sero-reaction, and the absence of the estivo-autumnal parasite in the blood will establish the diagnosis.

Prognosis.—Uncomplicated primary attacks of typical malaria usually recover under a proper and thorough treatment. Residence in a malarial district is always unfavorable though a very small percentage of the common intermittent variety will fail to respond to proper treatment. The pernicious types are least tractable under treatment and the previous health of the patient greatly influences the prognosis. The remittent form is exceedingly dangerous when it is complicated with typhoid. Uremia, hemorrhage and severe jaundice are unfavorable symptoms.

Treatment.—With our modern knowledge of the cause of malaria it is essential to give much attention to the prophylactic phase of treatment, for every malarial patient is a menace to a neighborhood. The same is true relative to the surrounding conditions, for every malarial district is a breeding ground for the mosquito. Therefore, prophylaxis contemplates not alone the isolation of the patient but every modern means for destroying the mosquito as well.

It is certainly fortunate that recent therapeutic investigation has established the fact that *quinin* certainly is a specific in this disease, inasmuch as it acts directly upon the intracorpuscular hematozoa. There seems to be, at present, no satisfactory reason for rejecting this theory of treatment when the results are so clearly demonstrated and no ascertainable harm has been found from using this remedy. While there is no doubt that other remedies are indicated for intercurrent symptoms and conditions we should not disregard the efficacy of this agent. The time for its administration is immediately after the paroxysm or at least after the sweating period. There is a divergence of opinion as to the amount required in each dose, some believing that the total amount for the day should be given in one or two doses a short time before the succeeding paroxysm. The object of this procedure is to surcharge the blood with the drug about the time the hematozoa sporulate. One of the objections to this procedure is the fact that many patients are thus liable to suffer from the toxic effects of the drug which would be indicated by nausea, deafness and tinnitus. Thus the consensus of experience seems to favor the divided doses amounting in all to sixteen to thirty grains per day. In case there is no response from this dosage it is permissible to increase the amount to at least forty grains daily. It is often best to give the largest dose only a few hours before the approaching paroxysm. When the paroxysms are less frequent and severe the dosage should be gradually reduced. For the sake of more prompt gastric absorption a few drops of dilute hydrochloric acid may be given after each dose unless the patient gives evidence of a sufficient amount of this in the stomach. The remedy may be introduced by means of the enema or suppositories when there is absolute disturbance from its introduction into the stomach. The hydrobromate of quinin has been successfully employed hypodermatically, the dose being fifteen grains, but abscesses usually follow.

The results of the quinin treatment will be observed by the prompt or gradual relief of the paroxysms and the disappearance of the plasmodium in the blood. Other preparations of cinchona, such as cinchonin, chinoidin, and the sulphate of quinidin have been recommended but are not so popular. *Arsenic* has been successfully used alone or in conjunction with quinin and is said to give best results when increased to its physiological effect. *Methylene blue,* in one to three grain doses four times daily, has been substituted for quinin but with questionable results. It may be used when quinin is contraindicated. I must confess that my early teaching influenced me unfavorably regarding the use of quinin and when I did accept its indication I was inclined to the minor dosage, generally using the first or third decimal potency. I am now satisfied that a small dose is not sufficient and this is easily confirmed when the blood is properly and regularly examined. The error in claiming too much for the minute dose is often due to a faulty diagnosis. To my own satisfaction I demonstrated the necessity of full doses of quinin in the treatment of these cases when I had the opportunity of treating many of our soldiers who contracted the disease during the recent Spanish-American war. I faithfully tried the lower potencies but with no results and the same was true of other remedies while almost immediate results were obtained by full doses of quinin. In this experience methylene blue also failed to give the satisfaction expected of it.

During the *paroxysm* every effort should be made to keep the patient warm by a sufficient amount of clothing. The paroxysm is often, however, so severe that this will almost be impossible. It will many times require the services of several strong attendants to keep the patient in bed. When the sweating stage intervenes the clothing may be gradually removed and the tepid sponge with alcohol and water is often conducive to comfort. So far as possible the patient should then be kept quiet and encouraged to sleep. During the *interparoxysmal state,* at least after the sweating stage has passed, it is not absolutely necessary to keep the patient in bed though too much liberty must not be permitted. The diet should be nourishing, easily digestible and decidedly limited in amount.

In the *pernicious intermittent* it is necessary to get the quinin effect as soon as possible. For that reason it has been recommended to administer the drug hypodermatically. Osler advises the use of the bisulphid in thirty grain doses, with the addition of five grains of tartaric acid, every two or three hours. Intravenous administration of the soluble bimuriate is also recommended; fifteen grains of this with a grain of sodium chlorid in two drams of distilled water may be injected. Stimulation is often required in these extreme cases. Aside from the regular indicated remedies the gastro-intestinal disturbances will call for careful dietetic and other adjuvant palliation. The restlessness and other nervous symptoms will demand care in accordance with previous suggestions for such conditions. During the apyrexia the administration of quinin must be pushed in order to avoid the severity of a recurring paroxysm.

The *remittent fevers* will necessitate a very different plan of treatment, inasmuch as they assume the typhoid character. The diet must be absolutely liquid, the gastro-intestinal disturbance must be relieved by appropriate remedies and enemata, the febrile symptoms will often demand the sponge bath and the usually indicated remedies. Quinin is, however, of specific value and must be administered in full doses. In severe cases with tympanites and other typhoid symptoms it may be necessary to stop all food for a time and resort to temporary nutrient enemata. A small dose of pilocarpin (gr. 1/8 to 1/6) has been advised as a hypodermatic medication when the fever is extremely high. The unfortunate depression of the heart, however, usually contraindicates this. *Malarial hematuria* will call for a limitation of the dosage of quinin even though this drug is necessary to overcome the parasites. Some authorities claim that it is contraindicated in this form of malaria but that is probably not true. In the milder forms of hematuria it is probably the best procedure to limit the amount at each dose. We shall probably obtain better results from the internal administration of such remedies as *hamamelis, ergotin, terebinth, mercurius, ferrum phos., crotalus* and *adrenalin.*

The homeopathic remedies will have a sphere of usefulness independent of the specific use of quinin as they will be required to meet the totality of symptoms pertaining to the peculiar idiosyncrasies of each case. It is not alone necessary to destroy the parasite with quinin but the ravages of its infection will surely leave a depreciating influence which will be reflected in symptoms and must be treated according to symptomatic suggestions. While I believe in the specific use of quinin I do not believe that this is all that is required in the treatment of malaria any more than one remedy or one dose of a remedy applies alone in the treatment of any disease. To practice medicine scientifically and successfully requires a broad-mindedness to interpret the utility of a principle of practice and at the same time accept the value of specific medication.

The *fever* of malaria may be attended by many symptoms which quinin alone probably does not arrest even though it destroys the cause. There may be many latent systemic conditions which this parasite arouses by its toxic effect. These then will require a careful investigation of their pathological meaning and, therefore, the affiliated remedy will be needed. *Aconite* and *veratrum viride* will often be demanded when there is evidence of high arterial excitement, restlessness, anxiety and dry surfaces. On the other hand *belladonna* may be indicated when there is a full and rapid pulse with throbbing carotids and the moist and flushed surfaces. The peculiar mental features of this remedy may also be reflected in the delirium and mental excitement so characteristic of its action. The dilated pupils, the convulsive action of the facial muscles, the dryness of the fauces, the painful and swollen joints, and the sudden changeableness of all its symptoms together with many other positive indications for this remedy will surely suggest its usefulness. Compared with this we must always take into consideration such remedies as *hyoscyamus, stramonium* and *chamomilla.* When prostration is pronounced, when the surfaces are cold and muscular weakness is pre-eminent then we must look for other symptoms which confirm the usefulness of *gelsemium.* In conjunction with this remedy it will frequently be necessary. to compare the symptoms of *baptisia, cimicifuga, echinacea* and *bryonia.* In the typical intermittent we shall find that the characteristics of *cedron* are frequently presented. The neuralgic pains, starting usually in the supraorbital area, are reflected over the head and to a certain extent over the whole body. Lancinating pains are felt also in all the joints while a peculiar numbness seems to pervade the whole body indicating thereby a profound depression from an extreme toxic agent. The same asthenic conditions will also call attention to *lachesis, naja* and *rhus tox.* When the symptoms seem to have reference to traumatic effects it will be natural to consider *arnica, bellis perennis, hamamelis* and *calendula* while *hypericum* will invariably meet pains which follow from nerve injury or pressure.

Eupatorium perfoliatum has so long been used in the form of "boneset tea" for all conditions which simulate the malarial fever that it would seem natural to think of this remedy in the treatment of true malaria. While it seems to apply more especially to the constitutional conditions which pertain in the chronic state of malarial cachexia it still has many symptoms of value in the acute stage. The peculiar periodicity, the bone pains, the occipital headache and the aching pain in the back are symptoms worthy of careful consideration. The febrile conditions are also suggestive as the chill appears early in the forenoon and is preceded by unquenchable thirst. On account of its gastric and hepatic influence it is frequently compared with *bryonia* and *chelidonium.*

In the *typhoid condition* it will be a good therapeutic principle to call into service all of the requirements outlined under the treatment of that disease. For that reason we shall always find it best to treat the disease with some reference to typhoid fever. Thus *baptisia, bryonia, belladonna* and similar remedies will be required. In the algid variety *veratrum album* and *camphor* will often be indicated, while in choleraic conditions we may add to these *cuprum, arsenic* and the *arsenite of copper. Methylene blue* is also of inestimable service in this form of the disease.

For the *chronic malarial* condition there are many remedies which will be required according to their therapeutic suggestions. *Arsenic* seems to be most essential as it relates so clearly to the anemic state and the secondary conditions of the malarial toxemia. This may be used in many forms though *china ars.,* and *arsenicum alb.,* are probably the most serviceable preparations. *Arsenite of iron* and the *arsenate of strychnia* are often of service and in all probability *arsenicum iodid* is occasionally indicated. *Ferrum phos., ferrum iodid* and many of the blood iron preparations may be considered with favor in many cases. As the fever subsides and the other characteristics of the malarial paroxysms disappear other remedies which are useful in combating the systemic depletion will be demanded by the prevalence of special symptoms. The diet should now be a rigorous one and a sufficient out-door life should be sought to build up the debilitated condition of the patient.

DYSENTERY.

This is an infectious inflammation of the large intestine embracing several varieties which, in the acute form, are clinically characterized by pain, frequent discharges from the bowel of mucus and blood and the attending symptoms of tenesmus, fever and prostration. In the chronic form diarrhea, alternating with constipation, is the chief symptom. Ulceration of the intestinal mucosa is the principal anatomical lesion. The disease may be studied under three distinct varieties, catarrhal, bacillary and amebic dysentery.

Catarrhal Dysentery.—This is the simplest form of the disease and it is commonly observed in the temperate climates. The mucous secretion of the intestine is augmented, the epithelium is exfoliated, while the solitary lymphatic glands of the large intestine and the solitary and agminated glands of the small intestine are also involved. In the milder forms there is only a swollen and congested condition of the mucosa with necrosis and small ulcerations, while in the severer cases the membrane is covered with mucus and streaked with blood, the entire mucosa being involved in a purulent inflammation with more extensive ulceration. The mucosa presents minute bleeding points and ecchymoses, while the lymphoid nodules are large and prominent. Ulceration may be discovered about the lymphoid structure to a limited extent. The *etiological factors* are not many. Season is most prominent, most cases being found in the summer and autumn. *Previous diseases* which leave the intestinal mucosa impaired and therefore open to new infections, are important causes. Thus typhoid fever and tuberculosis may be contributing causes and to a certain extent it may follow the acute exanthemata. It is often the result of such intestinal irritants as unripe fruit and indigestible food. In such instances the ileum is also implicated and the disease is in reality a form of entero-colitis. The bacillus of Shiga is often found in many cases.

The *symptoms* are characterized at first by the occurrence of a painless diarrhea with copious stools. Very soon these change to small mucous discharges which are streaked with blood. The number of evacuations varies from ten to one hundred daily. Cramping abdominal pains (tormina) then ensue and with these there is much complaint of tenesmus. The last is the most distressing symptom of all as it creates a very disagreeable sensation of rectal incontinence. A *chill* may be an initial symptom and fever, at first slight, though in some cases reaching 103° to 104° F., is an unfailing feature. The *pulse* is somewhat accelerated while thirst is always present and is sometimes extreme. The *tongue* is coated, moist at first and later very dry and red. *Nausea* and *vomiting* are occasionally distressing symptoms. The *abdomen* may be tender and tympanitic but in many instances is flat and hard. The *stools* often present various characteristics, sometimes containing hard scybala and at other times having a bilious appearance on account of the increased amount of bile. The microscope will also detect the presence of blood-corpuscles, leukocytes and oval epithelioid cells containing fat-drops and vacuoles. Usually no specific organisms are discovered though the bacillus pyocyaneus is claimed as a factor. The *duration* of the average case is not long as it usually terminates in a week under the appropriate diet and treatment. In exceptional cases it may resist the most thorough treatment and be prolonged into a chronic stage. The *diagnosis* will be easily made when the characteristic symptoms and the blood-stained stools are recognized. *Malignant diseases* of the rectum must be excluded by a careful differentiation. The *prognosis* is usually favorable.

Bacillary Dysentery.—This may appear in a variety of forms and is followed by a secondary ulceration or it may assume a chronic condition of long standing. It is most frequently found in temperate or tropical regions, an epidemic form being of common occurrence in Japan. It has also prevailed in asylums and camps, the aggregation of people favoring its development. The lower part of the large intestine is the usual seat of the lesion though it may invade the entire large intestine, occasionally involving the lower part of the small intestine as well. The *bacillus dysenteriae* is without doubt the cause and this may be defined as a micro-organism belonging to the colon typhoid group of bacilli. Its morphology is slightly different from the typhoid bacillus. Laboratory experiments have been made by injecting the organisms into the intestine of cats or by feeding these animals with them after alkalization of the gastric juice, thus establishing their positive action upon the large intestine. When taken into the digestive tract of man a severe colitis is rapidly established. Bacteriological diagnosis may be instituted by the recovery of the specific organism from the stools and by obtaining an agglutination reaction similar to the Widal test. The reaction will be established about the third or fourth day with dilutions varying from 1-20 to 1-1000 in a period of from one-half to one hour.

The *morbid anatomy* will present various characteristics. In the acute cases the entire large intestine and part of the small intestine is involved, the mucosa being greatly swollen and overspread with serum and blood. A fatal termination usually takes place in two or three days in this form. The pseudo-membranous is fortunately a more common form and in some instances this may take on the diphtheritic characteristics. In this the lesion may be located in different parts of the large intestine, or in the sigmoid or rectum. The membrane appears at first upon the elevations of the valvuli conniventes and later the intervening mucosa is covered with it. The mucous membrane is injected and small bleeding points may be observed beneath the mucus covering it. The histological structure of the exudate is both fibrinous and cellular, The glands of Lieberkühn are also affected and frequently undergo a necrosis while the blood-vessels of the mucosa are often occluded by thrombi. As exfoliation takes place dysenteric ulcers are often a sequence and the necrosis with its attending inflammatory changes may extend to the submucosa or terminate at this stage. The process of repair which naturally follows leads to a thickening of the intestinal wall which may be permanent. The appearance of secondary micro-organisms may protract the ulceration which, extending into the deeper tissues, may set up a chronic ulcerative dysentery. As connective tissue formation naturally follows a narrowing of the lumen may lead to serious contractures and deformities. As the inflammation may extend to the peritoneal coat adhesions are liable to be serious sequelae and perforation may rarely result.

The *symptoms* of this form of dysentery are similar to those of the catarrhal variety though they are more intense in character. The temperature reaches a higher degree, pain is much more pronounced, while the tormina and tenesmus are more distressing. The stools are distinctly bloody, delirium is pronounced and the adynamic condition is extreme. The abdominal tenderness and distension are marked and in some instances the symptoms assume a typhoid character. The *complications* are often numerous and varied. Abscess of the liver is one of the common ones as it may arise from thrombosis or embolism from the bowel into the portal system. When perforation of the bowel is a complication it will give rise to a localized or general peritonitis, more often the latter, which is usually fatal. Pleurisy, pericarditis, endocarditis, anasarca, arthritis, phlebitis and nephritis are also quite common sequelae. The *diagnosis* rests upon the distinctive symptoms and the bacteriological examination. The prognosis is always unfavorable as the toxemia tends to develop a fatal asthenia.

Amebic Dysentery.—This is an ulcerative inflammation of the large intestine found in both the temperate and the tropical countries. It is caused by the ameba coli, an organism "varying from fifteen to twenty microns in diameter and is actively mobile when examined in the living state. It consists of two portions, an outer ectosarc and an inner endosarc. Its movements are brought about through the propulsion of the former, after which the granular inner substance flows into the pseudopedia. The ameba is phagocytic, taking up foreign substances from the intestine, etc., and especially englobing the red blood corpuscles. At present two varieties of ameba are distinguished as occurring in the stools, the first non-pathogenic—ameba coli mitis—and the second pathogenic—ameba coli. The former has been found repeatedly in healthy stools and it does not exhibit phagocytic properties for red corpuscles. In this country amebic dysentery has been found to occur as a sporadic disease, especially in the Southern States, and in some of the Northern and New England States as well." (Tyson.)

The *lesion* is found in the large intestine and sometimes in the lower part of the ileum and it is characterized by an ulceration which involves both the mucosa and the submucosa. In rare instances this process may extend into the muscular and the peritoneal coats. Amebae are found in the affected portions of the mucosa and in the necrotic tissue adjoining. The proliferation of connective tissue induces tissue changes in the intestinal structure similar to those in the bacillary form and frequently this process may tend to restore the normal condition of the intestine. A pseudo-membrane is never present unless there is a pathological combination with the bacillary dysentery. The ameba may be discovered in the stools and in the secondary abscesses of the liver.

The *symptoms* are similar to those of catarrhal dysentery only they are more prolonged and less clearly defined. The onset is not so sudden, the stools are not so frequent but are more inclined to be copious and liquid. The amebae are found in great abundance in the dejecta. The straining at stool is not so severe nor are the passages so numerous, and there are intermissions in which improvement is apparent, only to be followed by the usual symptoms. The fever is not usually so severe but is liable to manifest acute exacerbations. The *complications* are characterized by hepatic abscesses which may be single or multiple, the walls of which are ragged from the projections of necrotic tissue. A zone of cellular infiltration is found next to the innermost necrotic zone and this may destroy the neighboring liver cells by compression, while external to this there is usually a zone of intense hyperemia. Pus cells, however, are not very abundant in the inflammatory infiltration, fatty and granular debris together with the amebae forming the greater part of the abscess contents. The whole hepatic structure may be invaded by circumscribed necrotic patches. The abscess may rupture into the lungs and the amebae will be found in the expectoration.

The *diagnosis* will be easily made when the amebae are found in the stools. The *prognosis* is not generally favorable to recovery as the disease has a prolonged duration. The exhaustion is so pronounced that the patient is not liable to survive the removal of the cause. In favorable cases the disease usually lasts about four or six weeks.

Chronic Dysentery.—The chronic stage of dysentery may follow any of the varieties though it is of more frequent occurrence in the bacillary form. The lesions which characterize the acute stage are liable to be present, in a modified degree, in the chronic form. Ulceration in its various stages of improvement, is a distinct pathological characteristic though chronic dysentery may exist without any evidence of this morbid condition. The intestinal coats, particularly the submucosa and the muscularis, are usually thickened while the signs of blood extravasation and disintegration are evidenced by the dark-gray patches. Cicatricial contractions, cystic degenerations and polypus formations are not uncommon.

The General Treatment.—Under the general treatment it will be necessary to give due thought to the limitation of the epidemic. Patients should be isolated and the stools should be carefully disinfected. The diet also should receive the most careful attention lest an impure or an imperfect food shall favor a fatal outcome, or the protraction of the disease. Liquid food is essential; milk should be boiled or peptonized while broths and beef juice with well cooked barley and rice added are of great value.

In catarrhal dysentery it may be well in the early stages to empty the intestines by means of a mild cathartic like castor oil, and for the relief of pain ten to twenty drops of laudanum may be added. *Salines* may be of service in some cases unless there is serious depletion. Grain doses of *opium* are standard expedients for the relief of excessive pain, though this may often be accomplished by the indicated remedy. *Bismuth subnitrate,* in 10 grain doses, is often added, while the astringent action of *lead acetate,* in 1 to 2 grain doses is also of some service in this respect. *Suppositories* of opium have been successfully used, when the rectal irritation indicates serious involvement of the lower bowel. *Camphor mixtures* with additional doses of opium have been popular expedients for the prompt palliation of pain and the relief of excessive diarrhea with cramps and tenesmus. That this is not a proper procedure in every case we all must admit.

In the *bacillary dysentery* great care should be exercised to avoid the irritating foods and to employ a form of nourishment which leaves as little residue as possible. Peptonized foods, beef peptonoids and beef juice will be efficacious in this respect while it will always be best to give small and frequent feedings. *Opium* is usually required to relieve the severe pain and in many cases the hypodermatic use of morphin will be demanded. The object is also to restrain the peristaltic excitement as well as to give relief from pain. The internal use of ***bismuth,*** in large doses, and ***silver nitrate,*** in small doses, have been recommended, but it is questionable how much relief is obtained by these medicinal agents. Tyson advises the employment of *iodoform* in 1/2 grain to 3 grain capsules three or four times daily.

The *amebic form* of dysentery will require a limited diet the same as in the other varieties. The indicated astringent and antiseptic remedies—particularly the sulphocarbolates—may be successfully employed. The use of *ipecac* has received much attention from the East Indian physicians, who have had extended experience in this form of the disease. It is supposed to act as a muscular sedative and a secretory stimulant. It thus stimulates peristalsis and augments the bile formation which is a natural intestinal antiseptic. Heroic doses —20 to 60 grains—are suggested for the first day, no food being allowed immediately before or after its administration, ice only being tolerated to quench the thirst. A much smaller dose is given on the second day and bismuth or opium may be added to relieve pain and quiet the peristaltic excitement. Milk is then prescribed in very small quantities and later farinaceous foods and soups may be cautiously given. Aside from the normal action of ipecac it is supposed to have a destructive influence upon the ameba. Whether this will supplant the minor doses of this remedy can be answered only by those who have had extensive experience with this disease. Osler recommends warm injections of quinin (1 to 5,000, 1 to 2,500 or 1 to 1,000). *Dysenteric vaccines* and antidysenteric sera have received some attention as they are supposed to have protective and curative properties. Conclusive evidence as to their efficacy is as yet wanting.

In the *chronic forms* of dysentery the same consideration for diet must be carried out though it is often possible to allow a little more liberty in this respect. The remedies mentioned for the relief of pain and peristaltic excitement are also to be considered but the best results have been obtained from the topical treatment. In many instances this may also be applied successfully in many of the acute conditions. When tenesmus and rectal pain are distressing these may be contraindicated. The patient is placed in the dorsal position with a pillow underneath the hips to favor gravitation of the medicated fluid high into the colon. Water at the temperature of

100° F. is then allowed to enter the bowel slowly through a colon tube. *Various astringents* such as lead, zinc and copper sulphate, and the nitrate of silver are added to the solution. The latter has received most favorable commendation and it has been advised in large doses—20 to 30 grains to the pint of water. Much smaller doses are, however, required in the early treatments. It is questionable how much permanent relief may be obtained in this way and in many respects the treatment should always be undertaken with the knowledge that it may cause secondary irritation. It is possible that warm water alone will suffice and it is safe to assume that any topical treatment which causes aggravation should not be continued.

The *internal medication* demands most favorable consideration. *Mercurius corrosivus* will naturally be thought of first and it certainly applies in the majority of cases. The frequent and scanty stools, their bloody and mucous character, the tormina and the tenesmus are all characteristic suggestions for this remedy. In the majority of cases it will accomplish more than all the topical treatments and the sedative action of drugs. *Mercurius sol,* is claimed to act better in the sporadic dysentery than in the malignant forms. *Mercurius dulcis* is probably more useful in the milder cases when pain and tenesmus are not so pronounced. *Arsenicum alb.* will no doubt be indicated in a fair proportion of cases, particularly when the stools are excoriating, offensive in odor, scanty in character, together with the symptoms of thirst, exhaustion and restlessness which usually characterize the symptomatology of this remedy. *Ipecac,* in the lower potencies, will probably be more efficacious than in the tincture. It will serve to allay the nausea and vomiting and at the same time correct the frequent green stools. It is also indicated when errors of diet have been the exciting cause. *Nitric acid* internally is often a better expedient than heroic doses of the same in the topical applications. It is suggested in the subacute and chronic forms when pus discharges are associated with the bloody mucous dejections. It is also useful in the typhoid type of the disease and when the discharge gives evidence of a diphtheritic formation on the intestinal mucosa. *Sulphur* in the true chronic case, may sometimes be of more service than any other remedy. It refers to the blood-streaked mucus, the constant tenesmus, the presence of a probable dyscrasia and the aggravation of symptoms at night or early in the morning.

There are three other remedies which seem to have a relation to a certain set of symptoms. *Colocynth* will naturally be thought of first when the agonizing abdominal pain is experienced in the early attacks of the acute form. The patient attains relief only by pressure in bending over, and the same cramp-like pains are inclined to extend to the limbs, while the dysenteric attacks are invariably aggravated by the least food or drink. *Dioscorea, chamomilla, cocculus* and *plumbum* may be compared with this remedy. *Cantharis* has a similar symptomatology in some respects, but it pertains to a more general extension of the inflammatory action, the urinary tract being also affected. The stools are like intestinal scrapings and are watery and bloody in consistency; violent burning pain extends throughout the intestinal tract after each bowel movement and urination is frequent and painful. *Apis, cannabis sativa* and *pulsatilla* may be compared. *Capsicum* has similar symptoms though the abdominal pains are not so pronounced; abdominal distension is often extreme, blood is more in evidence and thirst is characteristic. *Cuprum metallicum* is naturally associated with the above three remedies, though the spasmodic action may take on the convulsive form and a general muscular involvement is recognized; coldness of the extremities is a prominent symptom, thus indicating a circulatory disturbance following the depletion from diarrhea. *Cuprum arsenite* is possibly a better form of copper for the violent abdominal pain. *Veratrum alb.,* which has reference to more extreme coldness, perspiration on the forehead with vomiting and purging, *agaricus,* which is characterized by the addition of gastric cramps, and *camphor,* which represents a more complete choleraic collapse, should be studied under the same line of symptomatology. *Aloes* deserves special attention when the rectal symptoms are aggravated by hepatic disorder and portal congestion. It will be suggested when rectal pain, burning and incontinence are disturbing while the abdomen presents the signs of a distressing distension. *Alumina* and *lycopodium* may be compared. *Opium,* in potency, will often accomplish more by its internal administration than is obtained by the sedative action of heroic doses. The great danger resulting from the use of this drug to check the bowel action can not be sufficiently accentuated.

The *febrile* condition can not be considered without due reference to *aconite,* which presents a symptomatology similar to the early stages of dysentery or to *belladonna,* which represents many of the later phases of the fever when the nervous symptoms are marked, or *baptisia* and *rhus tox.* when the typhoid features are pronounced. It is also necessary when the signs of exhaustion are manifested to give due thought to *arsenic, china, ferrum phos., ferrum picricum, zincum phos., nux vomica* and many similar remedies.

CHOLERA.

Many terms have been applied to this disease. Cholera Asiatica has reference to its most frequent location. Cholera algida refers to its characteristic symptom, cholera infectiosa denominates its specific cause, while cholera maligna pertains to the serious phase of its epidemic occurrence. It may be defined as an acute infectious disease caused by the comma bacillus or spirillum of Koch. It is clinically characterized by its epidemic occurrence and by the vomiting, purging, painful cramps and the symptoms of collapse which attend it. There have been periodic visitations of this disease in many parts of Europe after an endemic prevalence of long standing in India. It usually appears in the epidemic form, and it made its way into the United States as early as 1832, and a second visitation occurred in the years 1835 and 1836. In 1854 a severe epidemic again prevailed in this country and a less extensive one reached our shores again in 1866. Since that time we have, with the exception of a few sporadic cases, been exempt from its affliction. As late, however, as 1892 a serious invasion afflicted the northwestern provinces of India and from thence it spread through European Russia and Prussia.

Etiology.—The *specific cause* of cholera is the spirillum of Koch, which is found in the intestinal canal of patients suffering with this disease, associated with the bacillus coli communis and other bacteria. It is a slightly curved rod, often assuming the shape of a letter S and half as long as the tubercle bacillus but somewhat thicker. It is motile and has been classed among the spirochetae. Its multiplication is favored by heat, moisture and filth, but it is destroyed by acids and by a temperature of 140° F. The stools of an infected patient will yield the cholera spirilla in great numbers, while their presence in the vomitus is rare. They have been repeatedly found in drinking water. *Predisposing causes* are found in the soil humidity and porosity and also in the warmer climates, and thus it is more common during the warm season. Age and sex have no predisposing influences, though the lack of proper sanitation is favorable to its propagation.

The *media of infection* are drinking water and contaminated food, though the spirilla are supposed to be conveyed by the hands or the clothing, and some observers claim that they may enter the mouth while floating in the air, but this, however, seems improbable. The disease is frequently disseminated by emigrants and makes its first impression near the seashore, a low altitude favoring its development. Insects are also considered as factors in the infectious contamination of articles of food. Pettenkofer claims that the spirilla which are found in the evacuations of cholera patients "must enter an appropriate soil and there undergo further development before becoming truly pathogenic." Enfeebled digestion, general ill health, alcoholic excess and nervous depression have much to do with the favorable reception and development of the spirilla. Immunity is not obtained by one attack.

Pathology.—*Emaciation* characterizes the general appearance. The skin has an ashen hue in the non-dependent parts, while there is a livid appearance of the dependent portions. The tissues are dry, inasmuch as the body has been drained of its liquids. A post mortem rise of temperature often takes place and muscular contractions may continue for some time after rigor mortis comes on. The *visceral lesions* are found chiefly in the intestines. In the early stage the serous part of the small intestine is congested, the muscular coat is relaxed, while the mucous membrane is injected, swollen and the seat of a fibrinous exudate. Very soon the stomach and intestines are filled with a "rice water" liquid with which there may be mingled a small amount of clotted blood. The solitary follicles and Peyer's patches are at first swollen, while later they may be the seat of ulceration. The post mortem will also reveal numerous ecchymotic spots in the intestinal mucosa which may be denuded of its epithelial lining. In case of death during the late stages of the disease patches of diphtheritic membrane may be found in the large intestine and other mucous surfaces. The bacilli are found here as well as in the dejecta.

The *stomach* and *esophagus* will often present a similar pathology; the *spleen* is small at first, but in the later stages of the disease it becomes larger and softer. The *liver* presents a passive hyperemia and cloudy swelling, while there may be signs of fatty degeneration and the desquamation of the epithelium of the cystic mucosa. The *kidneys* may not present any marked changes in the early stages, except a slight enlargement and some venous turgescence. Slight granular and hemorrhagic distintegration may be noticed in the cortex, but this is not common. Should death, however, take place late in the disease, the kidneys will exhibit a cloudy swelling and coägulation necrosis, while many other changes incidental to acute nephritis may be observed. The *heart* is not enlarged, but its walls are flaccid, the left ventricle is contracted and empty, while the right is filled with dark blood and soft clots. The *larynx,* trachea and bronchi are hyperemic, while the *lungs* are shrunken and small, being deprived of blood except in the dependent portions, which are in a hypostatic congestive state. The *brain* is hyperemic except when death occurs at a late period, and then its structure may be bloodless and edematous.

Symptoms.—The *period of incubation* lasts from thirty-six hours to four or five days, when the true symptoms of cholera present themselves. These are defined by three distinct stages, the preliminary diarrhea, the collapse and the reaction. *The stage of preliminary diarrhea* is characterized by a moderate diarrhea which may or may not be attended by colicky pain. The stools are yellowish in color and alkaline in reaction, nausea and vomiting do not exist, and there is slight evidence of depression with the exception of some headache and the intestinal discomfort. The temperature is rarely above normal. The first stage may continue for a week or it may last only a few hours.

The *stage of collapse* is characterized by a profuse diarrhea which has lost its yellowish color and assumed the "rice water" character. Muscular cramps now begin in the extremities and rapidly extend over the entire body, the abdomen being the seat of intense distress. Vomiting is now added to the diarrhea; at first this is bilious, but it soon assumes the rice water character, the discharges from the mouth and bowels becoming excessive. *Extreme exhaustion* now sets in, the face and eyes presenting a pinched expression, the countenance becoming pallid or bluish-gray. The entire body is cold and clammy, the superficial temperature falls five or six degrees, while the internal temperature may be as high as 103° or 104° F. The pulse is feeble, frequent and often absent. The heart sounds are not easily detected and the rapid respiration simulates impending death, as indeed the death coma may come on at any time. The secretions are all suppressed on account of the great loss of body fluids. In the vomitus and the bowel discharges the cholera bacillus and other bacteria may be found. In some instances the disease may be characterized by the lack of vomiting and purging, to which the term *cholera sicca* is applied. This stage of collapse is not generally long, being prolonged for twelve or twenty-four hours at the utmost. It may sometimes take the place of the primary stage of diarrhea. In all probability the severity of the collapse depends upon the liberation of the toxin by the bacilli at about this time.

The *stage of reaction* is represented by the return of warmth and color and the gradual establishment of the urinary and other secretions. The vomiting and purging are now less severe and frequent, gradually disappearing entirely. The diarrhea, however, may return unexpectedly with all of the attending symptoms of the second stage. Should this occur the patient is liable to die suddenly. Again, the *typhoid characteristics,* with an erythematous or roseolar eruption may appear, and all the stages of this condition must be passed through. *Nephritis* may be the terminating complication in other instances, or there may be a severe diphtheritic or catarrhal inflammation of the bowels. The secondary infection may also give rise to septic or pyemic invasions in other parts of the body. *Bronchitis,* pneumonia, erysipelas and parotitis are not infrequent complications.

Diagnosis.—The differentiations between a severe attack of cholera morbus and a sporadic case of true cholera or the light attack known as "cholerine" are often difficult to make. In *cholera morbus,* however, we may usually trace the irritating cause to an error in diet, the characteristic rice-water stools are not presented, the colicky pains do not extend in tonic cramps to the extremities, cyanosis, urinary suppression and the characteristic collapse are not recognized, and in addition we shall not find the spirilla in the stools. *Arsenic poisoning* and corrosive poisonings will be differentiated by the history and symptoms, while the gastro-enteritis, from this or other causes, will be recognized by the character of the stools, the absence of violent muscular cramps and the severe symptoms which follow from the great loss of fluid. If there is doubt, chemical tests should be made.

Prognosis.—The result of this disease depends upon its type and the vital resistance of the patient. The mortality varies in different epidemics, the percentage of deaths varying between twenty and eighty. Judicious and prompt treatment may save many cases. A prolonged epidemic seems to run its course, for in the last part of it there is an apparent tendency to a lighter form of the disease in which there is a larger percentage of recoveries.

Treatment.—The *prevention* of the disease has been almost attained in this country by the efficiency of quarantine protection. The same prophylaxis may obtain in each case, providing thorough disinfection is given to the vomitus and the stools. The nurse, the friends, and the surroundings must suffer the same quarantine and prophylactic care lest an epidemic be encouraged. Due care should also be given at this time to the water, the milk and the food that is used daily. All uncooked food should be rejected and no water or milk should be drunk unless sterilized. *Protective inoculation* has been obtained by the use of an antitoxic serum, but our acquaintance with that procedure in this country is somewhat limited. The prompt *treatment of the attack* has received special consideration by those who have been the closest observers in the management of the disease, the object being to "restrain the development of the bacilli in the intestine and to neutralize the cholera poison." Cantani suggested the injection into the intestine of 1/2 to 2 1/2 quarts of water, or infusion of chamomile containing 1 to 5 drams of tannic acid, 20 to 30 drops of laudanum and 5 to 12 drams of gum arabic. The temperature of the liquid should be at least 100° F. to avoid chilling the patient. These should be given three or four times daily and in severe cases after each evacuation. In the stage of serous diarrhea we should attempt to resist the watery waste by the hypodermatic use of morphin or the internal use of opium. *Bismuth, lead acetate, Dover's powder* and other astringents have been employed for this purpose. Cantani advocates the injection of an artificial serum containing 1 dram of sodium chlorid and 46 grains of sodium carbonate per quart of sterilized water warmed up to 104° F. into the subcutaneous connective tissue. This solution may be introduced through the cannula of an aspirator. Shakespeare recommends the performance of this treatment by means of "a fountain syringe with a long flexible tube furnished with a cock; with another shorter tube, one end attached to the cock, the other having a needle-pointed cannula, a little longer, stronger and with a somewhat wider caliber than the ordinary hypodermic needle. The tube and cannula are first perfectly filled with a fluid and then the cannula is inserted well in between the skin and deep fascia of the flanks, buttocks or interscapular region. The fluid should be made to flow slowly, allowing fifteen to twenty minutes for the introduction of one quart."

The *vomiting* may be relieved by ice, lavage or cold external applications. Heat may be a necessary external application for the sake of the surface coldness, and warm baths are often advised. For the same reason *stimulants* must be administered hypodermatically and of these whiskey, ammonia, strychnia and digitalis are frequently employed. For the *algid stage* hypodermatic stimulation, enteroclysis, hypodermoclysis and the external application of heat are all indicated. Saline intravenous injections are also demanded. The *stage of reaction* may require the continuance of many of these measures for some time and the symptomatic treatment.

Among the homeopathic remedies we shall find *camphor* most frequently indicated. It seems to correspond closely to the leading features of the disease and we have many favorable reports from the use of this remedy by practicians of our school. It should be employed in the lower potencies or in drop doses of the tincture on lumps of sugar. It has also been successfully used in the form of inunctions. *Ipecac, arsenicum, veratrum alb., cuprum, cantharis, terebinth, rhus tox, phos. acid, merc. corr.,* and *secale* are important homeopathic remedies. *Hydrocyanic acid* in the sixth to the second decimal potency, has given splendid results in the practice of Dr. Sivicar, of India, in the stage of collapse when the patient was apparently past help.

YELLOW FEVER.

Yellow fever is an acute infectious disease usually found in tropical and subtropical countries and is characterized by a febrile paroxysm which is followed by a brief remission and then a relapse. It is clinically recognized in the majority of cases by its symptoms of profound toxemia, jaundice and gastric hemorrhage or "black vomit." It may occur endemically or epidemically and it is probably caused by a specific organism which is transmitted by the bite of mosquitoes.

Etiology.—The disease, so far as we know, appeared first in the Barbadoes, West Indies, as early as 1647, and soon after in certain parts of the Spanish Main. Following the line of commercial extension it prevailed to a limited degree at different intervals in various European ports, never spreading very far from the seashore, and rarely attaining much headway in altitudes above 1,000 feet. It appeared in Boston about 1693 and since that time the worst epidemics have been limited to the seacoasts of the United States. In 1793 a serious epidemic occurred in Philadelphia and the coast towns of many of the neighboring states were visited by its scourge. A few years later it prevailed again in Boston and then visited Baltimore and other southern seaports. For the next few years there were only slight outbreaks when, in 1853, it raged severely throughout the southern States, and periodic epidemics have reappeared in these localities nearly every summer. A better knowledge of its predisposing causes and the general improvement in sanitary conditions has, without doubt, been a powerful factor in its limitations. Cuba has been its breeding ground, but since our recent war with Spain and the sanitary improvements in that locality, it is not so prevalent. Guitèras has classified the areas of infection as follows: (1) The focal zone in which the disease is never absent, including Havana, Vera Cruz, Rio de Janiero and other Spanish-American ports. (2) The perifocal zone or regions of periodic epidemics, including the ports of the tropical Atlantic in America and Africa. (3) The zone of accidental epidemics, between the parallels of 45° north and 35° south latitude.

Bacteriological investigations have not yet established the identity of the specific organism. Sanarelli described an organism as the "bacillus icterodes," but there has been no substantial confirmation in this respect. That the mosquito is the host of the unknown parasite was first claimed by Finley and a satisfactory confirmation of this deduction was established by Reed in 1900. His conclusions are as follows: "(1) The mosquito—Stegomyia fasciatus—is the intermediate host for the yellow fever parasite. (2) The fever is transmitted to the non-immune individual by the bite of a mosquito that has previously fed on the blood of those sick with this disease. (3) An interval of about twelve days or more after contamination appears to be necessary before the mosquito is capable of conveying the infection. (4) The bite of the mosquito at an earlier period after contamination does not appear to confer any immunity against a subsequent attack. (5) Yellow fever can also be experimentally produced by the subcutaneous injection of blood taken from the general circulation during the first and second days of the disease. (6) An attack of yellow fever, produced by the bite of a mosquito, confers immunity against the subsequent injection of the blood of an individual suffering from the non-experimental form of this disease. (7) The period of incubation has varied, in thirteen experimental cases, from forty-one hours to five days and seventeen hours. (8) Yellow fever is not conveyed by fomites and, hence, disinfection of articles of clothing, bedding or merchandise supposed to be contaminated by contact with those sick with the disease is unnecessary. (9) A house may be said to be infected with yellow fever only when there are present within its walls contaminated mosquitos capable of conveying the parasite of this disease. (10) The spread of yellow fever can be most effectually controlled by measures directed to the destruction of mosquitos and the protection of the sick and well against the bites of the insects. (11) While the mode of propagation of yellow fever has now been definitely determined, the specific cause of this disease remains to be discovered."

The above demonstrations of this investigator are given verbatim to call attention to the latest theory regarding the mode of infection in this fever, but it is safe to say that it is not yet fully established that the mosquito is the only means of the transmission of the unknown organism. We know also that freezing weather at least terminates the activity of the disease. Among the *predisposing causes* we must, therefore, regard the summer season as most favorable to its development. *Age* has some influence, for the young are most frequently afflicted. *Males* are said to be more predisposed and the negro, by his long residence in the locality where it prevails, is thought to be immune. One attack generally gives immunity against its recurrence.

Pathology.—*Jaundice* and subcutaneous hemorrhages are marked features of the pathology. The *blood serum* is red in color because it contains dissolved hemoglobin. The *liver* usually has a pale-yellow or a brownish-yellow color, while the cells are in various stages of fatty degeneration. In the early stages it may be hyperemic and enlarged. The gall bladder is generally empty. The *heart* has a pale color and granular or fatty degenerations are in evidence. The *kidneys* show signs of diffuse nephritis, the epithelium of the convoluted tubules is swollen and granular, fatty areas are often found at the base of the pyramids and various bacteria are present in abundance. The *spleen* is not enlarged, but has a friable consistency and a dark color. The "black vomit" is found in the stomach, the mucous membrane of which is swollen and studded with blood extravasations.

Symptoms.—*A period of incubation,* characterized by headache, malaise, anorexia, etc., usually precedes the initial symptoms by from one to five days. The *stage of invasion* then develops suddenly with a distinct chill and is soon followed by a fever in which the temperature rapidly rises to 103° or 105° F. The pulse is accelerated at first but may fall as the temperature goes higher. The *fever* is usually accompanied by severe headache, pain in the back and legs, while restlessness, mental confusion and occasional delirium are often attending symptoms. The *skin* is hot and dry, the face is flushed, the eyes are injected, while the eyelids and lips may present a light tumefaction. The *tongue* is moist, but coated, the bowels are constipated, the urine is scanty and very frequently albuminous. *Slight jaundice* usually appears at this time while nausea and vomiting—the vomitus resembling coffee—are nearly always present during this period. This stage lasts from a few hours to several days.

The *stage of remission,* or the stage of calm, follows the initial stage and this is often the beginning of convalescence in mild cases. It is characterized by a decline in the fever and a general improvement of all symptoms. Some signs of ill health such as prostration, gastric irritability, mental dulness and often a slight jaundice may persist. In severe cases this stage is short, lasting only a few hours or a few days.

The *stage of febrile reaction* then supervenes, lasting in general only two or three days. This is marked by a rising temperature though the pulse may continue to fall and profound collapse supervenes. The body surface is cold, the skin is distinctly yellow or bronzed, while nausea and vomiting return with renewed force. *Hemorrhage* into the stomach now takes place, giving rise to the peculiar "black vomit" which is such a typical feature of the disease. The stools may also become tarry as the result of intestinal bleeding, while hemorrhage from other mucous membranes, epistaxis, metrorrhagia, etc., may appear. *Cutaneous hemorrhages* are also common, the tongue becomes dry, brown or even black, and sordes collect on the teeth, simulating an extreme case of typhoid. *Albuminuria* is common, the strength fails rapidly, the urine is often suppressed, while the nervous excitement may terminate in convulsions or be followed by coma and death. On the contrary the extreme symptoms may gradually subside and recovery will take place, though the jaundice may continue for some time. Finley describes three varieties of the fever: The acclimation fever or non-albuminuric yellow fever, the plain albuminuric yellow fever, and the melano-albuminuric yellow fever, which is characterized by the presence of blood in the stomach or intestines.

Diagnosis.—Guitèras gives three typical symptoms which should guide us in making a diagnosis. These are the *facies,* including especially the early jaundice, the *early presence of albuminuria* and the *slowing of the pulse* with a steady or even rising temperature. When we add to these the peculiar characteristics of the initial stage and the stage of collapse, together with the intervening remission, it will not be extremely difficult to diagnose the disease. The serum diagnosis is also claimed as an important and practicable test, but as yet is not generally accepted. The *differential diagnosis* will concern several simulating fevers. The *malarial remittent* will be differentiated by its prolonged chill, its different temperature curve, the presence of Laveran's plasmodium, the vomitus with bile-pigment and bile-salts and the absence of early albuminuria. *Dengue* is sometimes very confusing, inasmuch as it so frequently coexists with epidemics of yellow fever and is prevalent in the same localities. The difference between the two diseases is not easily established as they have so many symptoms in common. In dengue, however, we shall not find the facies, the early albuminuria and the slowing of the pulse with the rise of fever which are so characteristic of yellow fever. Hemorrhages are also much less common and the jaundice, when present, rarely appears so early.

Prognosis.—The disease must always be regarded as severe and usually fatal. The improvement in sanitation and the general management of infectious diseases is certainly reducing the percentage of mortality. Prophylaxis we certainly have within our reach and in all probability the severe epidemics which have prevailed in the South will not occur again. The serious symptoms may be enumerated as the intense capillary congestion during the early stages, the suppression of urine, the intense jaundice and the uremic toxemia.

Treatment.—The *prophylactic* treatment of yellow fever will naturally take into consideration the protection of the sick against the bites of mosquitos and also the elimination of this pest by proper drainage. It is thoroughly settled that we shall be able to destroy many of the larvae by the use of petroleum. When a case of yellow fever occurs it is best that complete isolation shall be established and that the patient shall be completely screened so that no mosquitoes shall be able to spread the epidemic.

The *general management* of the fever will call into use all of the requirements of treatment for any severe fever. The patient should be placed in bed and kept very quiet in a room with sufficient fresh air without exposure to a draft. The nourishment should be liquid and the patient should be fed with a spoon or tube in order to avoid any undue exertion. In the early period no nourishment whatever is necessary. Cleanliness of bed linen and patient are mandatory requirements. So far as *medication* is concerned no specific or satisfactory line of remedies has been settled upon. *Elimination* must be encouraged and thus cathartics and diuretics are required. Castor oil is considered by some experienced physicians as the best expedient for the first and a sufficiency of pure water ought to attain the second object. *Hydrotherapy*, in a modified form, may be used to reduce the high fever, but this should be carefully employed previous to the decline of the temperature. Pain may require such remedies as acetanilid, phenacetin, ammonol and exalgin, in 3 to 5 grain doses every three or four hours. In larger doses these may have some antipyretic effect, but the danger of cardiac depression must always be guarded against. It is possible that we may get the same relief from *spigelia, bryonia, arnica* or *rhus tox.* Morphin or codein may be required for the temporary relief of excruciating pain, but these are liable to cause an unfavorable reaction in the elimination already attained. *Quinin,* though of little effect as compared to its use in malaria, is nevertheless of much importance in the relief of pain. *Intestinal antiseptics*—particularly the sulphocarbolates—are of much value. The normal salt enema is also a powerful adjuvant in this respect, and in case of extreme constipation a saline may be used. *Nutrient enemas* will be indicated in case of severe vomiting, and ice may be given by the mouth for some time. Sternberg advised the following: Sodium bicarbonate drams 4, bichlorid of mercury grain 1/2, and pure water, 1 pint. Of this mixture two teaspoonfuls every hour day and night for a severe case, and every hour by day and every two hours by night for a mild case, are to be given, the object being, by the use of this antacid and antiseptic, to restrain the development of any specific organism in the intestine. For hemorrhage *aconite, hamamelis, ipecac* and *nitric acid* may be employed. *Ergot, lead acetate* and *opium* may also be considered for the same purpose. The serum treatment has not yet proven satisfactory.

During the *period of remission* stimulation will be required to support the failing strength and to avert the cardiac exhaustion. For this purpose tonics, digitalis, strychnia or alcohol may suffice, though for the same purpose we may increase the food somewhat. In the stage of collapse the supportive measures will be in more active demand and it will be often necessary to increase the stimulants or to administer them hypodermatically. Rectal nourishment may be required throughout this stage.

When we take into consideration the prophylactic requirements so far as sanitation and the mosquito are concerned, it would seem that the force of our treatment must be applied in this respect rather than by the application of remedies. When, however, the fever is contracted, an opportunity will often appear for the use of homeopathic remedies. The fever will certainly be regulated by such remedies as *aconite, belladonna, baptisia* and *veratrum viride,* while the cold surfaces and the depression which attends a collapse will, aside from the stimulation, call for such remedies as *camphor, veratrum alb., arsenicum* and *ipecac.* These, of course, must be given in accordance with the totality of symptoms. The late Dr. Holcomb, of New Orleans, who certainly had a large experience with this disease when it raged severely in the South, has expressed a preference for *argentum nitricum, lachesis* and *crotalus.* He found them of especial value during the "black vomit" stage, and to these may be added *cuprum ars.,* and *arsenicum alb.* when the abdominal cramps are extreme. Goodno calls attention to *cadmium sulphuricum.* For the attending biliary symptoms *carduus, leptandrin, chionanthus, nux vomica* and *mercurius dulcis* may be mentioned. When the vomiting is extreme we shall be obliged to decide between *arsenicum, ipecac, tartar emetic* and *sulphuric acid. Cantharis* is often indicated when the urine is suppressed or its characteristic diarrhea is present. *Hyoscyamus, platinum* and *ammonium valerianate* may be of use when nervous symptoms are pronounced. *Phosphorus* would naturally be thought of with relation to the fatty degeneration of the liver and other organs.

DENGUE.

This is a disease which occurs most frequently in tropical countries and it is characterized by febrile paroxysms with extreme pain in the joints and muscles and the occasional appearance of a skin eruption. It has been known as "break bone fever" on account of the severe and deep-seated character of the pain and it has received its name from the expression "dandy fever," which refers to the stiff or dandified gait of those afflicted. The disease prevailed as early as 1779 in Cairo and in Java. Later it invaded the West Indies and certain parts of Spain. A mild epidemic appeared in Philadelphia one year later and several epidemics were experienced in our southern States in the early part of the nineteenth century. Since then the disease has been conspicuous on various occasions south of the thirty-second parallel of latitude, and in 1897 a severe epidemic raged in the vicinity of Galveston, Tex. Since that time it has not been so prevalent.

Etiology.—The etiological factors of this disease are not clearly established. McLaughlin, of Texas, discovered a micrococcus in the blood of a patient suffering with the fever, and Graham discovered an ameboid form in the red blood corpuscles which resembled the plasmodium malariae. The disease seems to spread in the wake of commerce, but has no natural habitat except in southern countries and it occurs generally during the summer months. Both sexes and all ages are affected alike and no definite morbid anatomical changes have been found. One attack seems to furnish immunity.

Symptoms.—After a *short incubation* period, three to five days, in which there is a marked sense of malaise, headache and slight chill, the *onset* begins with a distinct and severe pain in the joints and the muscles of the back and limbs, often being accompanied by excruciating pain in the head. The pain becomes so extreme that it seems to invade the osseous structure and hence the term "breakbone" fever. The *joints,* both large and small, are red, swollen and very tender. Coexistent with these symptoms the fever appears, the temperature ranging from 102° to 106° F., and reaching its maximum by the third or fourth day or sometimes sooner. The *pulse* is rapid, the *respiration* is decidedly quickened, while anorexia and other gastric symptoms are often distressing. *Thirst* is often extreme, constipation is usually obstinate and the urine is frequently scanty and may rarely contain albumin. The *face* is flushed, the conjunctivae are congested, hemorrhages from the mucous membranes are common and the "black vomit" has been observed in a few instances. *Delirium* is not a frequent symptom, though it may exist when children are afflicted.

The *initial fever* continues for three or four days, when it subsides by a marked remission. For a short period the patient is relieved of pain and convalescence seems apparent. *Prostration* takes the place of the early symptoms and during this time an *erythematous rash* is recognized. The *lymphatic glands* are frequently swollen and the indurations may persist for some time. A *second febrile* paroxysm, with a return of the characteristic symptoms and the appearance of a roseolar eruption may follow the remission. This, however, is generally milder in character and shorter in duration. A slow convalescence then ensues, though many of the symptoms may persist for a long time and another febrile paroxysm is liable to occur. Meningeal complications, catarrhal inflammations of the mucosa, pericarditis and many nervous disturbances may attend the extreme physical prostration of this stage, but are not common.

Diagnosis.—The diagnosis will be confused most frequently with *acute rheumatism,* but the epidemic character of dengue, its decided remission and its short duration should be guiding features for differentiation. Rheumatism shows no eruption or glandular swelling and the pain is confined mostly to the joints. *Influenza* has many symptoms in common, but the joints are not involved, the eruption is inclined to be herpetic and there is not a marked remission of the fever with a recurrence of this and the other symptoms. The presence of the influenza bacillus will settle the diagnosis. *Scarlet fever* is characterized by a continuous fever, the joints are not enlarged and the eruption is always erythematous. *Yellow fever,* having the same habitat, is often very confusing. In this the febrile rise is more regular, the fever has a longer duration and is not characterized by a remission with a secondary moderate rise. Further differentiating symptoms are as follows: In yellow fever the pulse falls with the febrile decline, eruption is not present, vomiting is frequent, albuminous urine is recognized early, jaundice is present early, hemorrhages are common and "black vomit" is characteristic; it is often a fatal disease and a second attack rarely occurs.

Prognosis.—Dengue will generally respond to good care and to the administration of remedies according to the symptomatic indications. Serious complications may arise and thus delay or complicate the cure, but fortunately these are not of common occurrence.

Treatment.—It is probable that many of the suggestions given under the subject of acute rheumatic fever will be required here. *Dietetic regulation,* rest and hydrotherapy have the same value in the treatment of this disease as in other infectious diseases. Relief from pain may demand specific medication, either hypodermatically or by the mouth. Otherwise remedies, employed according to their symptomatic suggestion, will be of most satisfactory service. In the febrile stage of the disease we shall no doubt find unusual demands in the early stages for *aconite* and *belladonna,* while the more profound depression attending it will call for *baptisia, echinacea, rhus tox, veratrum viride, gelsemium* and *cimicifuga.* *Eupatorium perfoliatum,* with its "breakbone" characteristics, probably applies more frequently than any other remedy. The relation of this remedy to the disturbance of the nasal, bronchial and gastric mucous membranes is equally suggestive, while the cephalalgic pains are distinctive. The febrile manifestations are the most important. The chill occurring in the forenoon, preceded by thirst and the great soreness in the bones, together with the relief by copious perspiration, are the most characteristic symptoms. It is reasonable to expect the greatest relief from its use in low potency and the tincture. *Bryonia* is another valuable remedy, as the pain is usually aggravated by motion and often assumes the peculiar stitching and tearing features of this remedy. It also covers the dry conditions of the mucous membranes, the mental irritability, the splitting headache and many of the gastric symptoms. *Conium, argentum nit.* and *camphor* may be compared. *Arnica* refers to a less intense muscular pain and a burning, itching eruption which tends to the formation of small boils. The eruption of *arsenicum* is more inclined to be papular, dry, scaly and burning; *rhus tox* has more of the erythematous or erysipelatous features; *urtica urens* pertains purely to the urticarial rash, while other remedies will often be suggested by their peculiar eruptive characteristics.

Spigelia refers to the neuralgias of the cranial nerves and the involvement of the joints of the upper extremities. It does not bear much relation to the febrile condition as there is a greater tendency to a prolongation of the chilliness. *Glonoin* (6x-12x) may often be required temporarily in the extreme congestive headaches, while *physostigma, hyoscyamus, anacardium* and *belladonna* may be applicable in many of the cerebral and nervous symptoms. The joint pains will many times furnish indications for *colchicin* and *sodium salicylate,* while *colocynth, plumbum* and *agaricin* will often meet the painful muscular spasms. Should hematemesis exist, *ipecac, secale, arsenicum* and *ferrum phos.* may naturally be considered and should the hemorrhage be of renal character, *cantharis, mercurius* and *terebinth* will be of service.

THE PLAGUE.

The term plague has reference to the Oriental disease which is generally recognized as the Bubonic Plague or "Black Death." It is characterized as an infectious disease in which there is a tendency to buboes or carbuncles and petechiae and high fever. It is extremely severe, runs a rapid course and generally occurs in epidemics.

Etiology.—Much of the history pertaining to this disease is probably confused with the appearance of typhoid and typhus. The bubonic plague has, however, been of long duration in certain portions of India, while European countries have suffered from its occasional epidemic appearance. Its outbreaks have usually been followed by numerous deaths and it has only been limited in its geographical distribution by the most vigorous quarantine. Since 1894 it appeared with terrific devastation in China and spread to Japan and Honolulu, while a few cases were observed in the Chinese section of San Francisco. The United States has been, for the most part, fairly exempt. During the epidemic in Hong Kong, through the efforts of Kitasato and Yersin, the specific bacillus was discovered and is described as "a short rod with rounded ends; it stains deeply at the ends, giving the appearance of a pair of micrococci; it is found in the blood glands and other viscera and pure cultures are readily made. It obtains entrance through the respiratory and digestive tract, but more especially by way of excoriations." The rat is supposed to be the most frequent carrier of the contagion, though it may be conveyed by man. Flies, fleas and ants are also believed to be agents of its transmission, while fomites may also be a means of its dissemination. It is essentially a disease of hot countries and hot seasons and unhygienic surroundings and therefore usually has its origin in the poorer quarters of a city. Cleanliness furnishes immunity and this is instanced by the rare affliction of those who are exposed when this precaution is properly taken.

Clinical Varieties.—*Pestis minor* is an abortive or larval form often appearing endemically before the real epidemic. In this there is only slight lymphatic swelling, the fever and constitutional symptoms are not marked and a favorable termination ensues in about two weeks. The *bubonic form* is the more common and typical form, inasmuch as the adenitis is most pronounced, occurring in about 70 per cent of cases. A *septicemic form,* also called toxic or fulminant, is always severe and is liable to terminate in death in twenty-four hours with associate hemorrhages; in this the glandular enlargement is not extreme, inasmuch as death takes place before the glands have time to swell. The *pneumonic form* is characterized by the development of the infectious process in the lungs instead of in the glands, the sputum containing numerous bacilli.

Symptoms.—The true bubonic form is characterized by a *period of incubation* lasting from two to seven days. This may be preceded, by a few hours or perhaps a day, by a period of *prodrome* in which headache, intense exhaustion, marked nausea, vomiting, vertigo and backache are prominent features. The physiognomy is stupid and the patient, suffering from the infectious intoxication, reels like a drunkard. The *invasion* is announced by a chilly sensation and sometimes by a pronounced chill. The *temperature* rises rapidly to 103°, 104° or even 106° F. The *pulse* is rapid, threadlike and often dicrotic. *Delirium* and *coma* are liable to supervene, the debility becomes more pronounced and *petechiae* or *vibices* may make their appearance. Albuminuria, hematuria and hematemesis may also be prominent symptoms, while there is occasional evidence of splenic enlargement.

The *bubo* or suppurating gland is the most typical symptom and this will be most commonly found in the inguinal regions, the axilla, the neck or the popliteal space. It usually attains the size of an egg and then is liable to rupture if not opened surgically. As suggested under the different varieties, the bubo may subside without suppuration, but that is always an unfavorable sign. The swellings are always excruciatingly tender before evacuation. A *profuse sweat* occurs when the bubo appears and the temperature may subside. Carbuncles often invade the tissue of the lower extremities or neck.

The *pneumonic form* will be characterized by a pronounced chill, high fever, severe localized pain in the chest, dyspnea and all the signs which attend the croupous exudate, together with unusual prostration. The *septicemic form* will be manifested by the evidence of a profound prostration, hemorrhages from the nose, bowel and kidneys and the slight though general glandular enlargement. Delirium, of the typhoid character, is usually recognized and apyrexia sometimes takes the place of the febrile action in the ordinary type.

Diagnosis.—The bubo and the carbuncle are the differentiating physical signs, while the other features of the disease are characterized by the fever, the intense prostration, the petechiae and vibices. The absence of the bubonic swellings will often obscure the disease. The bacteriological findings will confirm the diagnosis.

Prognosis.—Bubonic plague is an exceedingly fatal disease, the mortality being very high in each epidemic. Suppuration of the glands is regarded as a favorable symptom. The disappearance of the glandular swelling, the presence of pneumonia, intense toxic features, purpuric spots, carbuncles and hemorrhage are unfavorable symptoms. The average duration of fatal cases is from three to five days.

Treatment.—The treatment of the disease must have due regard first of all for the improvement of sanitary surroundings for general protection and the proper isolation of the patient. The excreta need even more antiseptic care than those of typhoid and after death the corpse should be cremated. Protective inoculation has done much to ward off the serious epidemics and the value of serum therapy is being demonstrated by Haffkine and Yersin. At present few practicians in the northern countries have had any opportunity to study this disease.

CEREBRO-SPINAL MENINGITIS.

Cerebro-spinal meningitis, also termed spotted fever, is an infectious disease caused by the micrococcus lanceolatus. It is characterized by an inflammatory invasion of the cerebro-spinal meninges and a somewhat irregular febrile course. It occurs most frequently in the epidemic form, though sporadic cases are occasionally found.

Etiology.—While in all probability this disease may have existed before 1805, it was not until that date that it received special recognition by Vieusseux of Geneva. Since that time it has prevailed in different parts of Europe and America, but epidemics have not been widespread or extensive. At present it is fortunately of rare occurrence. Country districts have been affected almost as frequently as the congested districts of the city, though it often becomes endemic in the latter. Soldiers have been occasionally afflicted and, therefore, camp life may be a contributing factor. Young adults and children furnish a majority of the cases; it prevails most commonly in the colder climates and is usually incident to the colder seasons of the year. Unhygienic influences predispose favorably, and it often follows from over exertion and depressing mental conditions. It has not as yet been conclusively settled how the contagion is conveyed to the patient, but the germ probably reaches the meninges by direct inflammatory extension or by blood metastasis. The disease is not regarded as of highly contagious character. Weichselbaum discovered the meningococcus or the diplococcus meningitidis within the polynuclear leukocyte as late as 1897 and his observations have been confirmed by experienced investigators since that time. Welch has claimed that this and the pneumococcus are varieties of the same bacterium, while Netter regards the meningococcus as a degenerate form of the pneumococcus. Mixed infections undoubtedly exist with the presence of these two microbes together with the streptococcus pyogenes, the staphylococcus aureus, the bacillus coli communis and the tubercle bacillus.

Pathology.—This is found principally in the brain and spinal cord, the meninges showing at first a distinct hyperemia which soon develops into an intense congestion. The final stage is characterized by the inflammatory products which are discovered underneath the pia mater. These are serous, fibrinous or purulent in character and are most abundant in the large fissures and sulci as well as at the base of the brain. The arachnoid spaces often contain both serum and pus. The convexity and deeper cerebral tissues may partake of the hyperemic condition and show some cell distintegration. The choroid plexus may give slight evidence of congestion, the ventricles are occasionally distended by the effusion and their mural structures are occasionally softened. The pathological changes are similar in both the brain and the cord, though in the latter the central canal may be filled with pus and the cord substance may be invaded by the degenerative process. Here also the exudate compression gives rise to local palsies or a distressing neuritis, while the irritation of the posterior roots causes intense pain. The diplococci are found in the inflammatory exudate and there may be a general infiltration of the brain and cord tissue with pus cells. As the disease progresses the meninges will show a marked thickening, varying in accordance with the character and extent of the exudate. The cranial nerves often suffer from a similar pathogenic invasion of their nuclei, the auditory and optic nerves being most frequently affected. Other organs present only slight pathologic changes. For instance a splenic enlargement may be detected when the fever is prolonged, the liver is often hyperemic and the kidneys are occasionally congested. The stomach and intestines are likewise similarly affected in some instances. The pneumonic process is more common inasmuch as the pneumococcus is so often a factor in this disease. Endocarditis is sometimes present while the pericardium and the pleurae are both liable to a serous or sero-purulent exudation.

Symptoms.—The symptoms will vary according to the character of the attack as the disease may assume various forms. In the ordinary form the period of *incubation,* if any exists, is of short duration, and prodromal symptoms are rarely recognized, though these may be present in the character of malaise, headache, gastric disturbances or premonitory pains in the neck, back and limbs. The *onset* is sudden and is usually attended by a marked chill which may amount to a convulsion in children. *Vomiting,* often of projectile character, soon ensues, though it does not generally last very long. It is not associated with any gastric disturbance as the tongue is usually clean unless typhoid symptoms intervene. The intestines are rarely disturbed as constipation is common and the abdomen may be distended or retracted. *Violent headache,* with pain in the back of the neck and the back, soon succeeds the vomiting and with this there may be more or less of myalgic pain throughout the body. *Hyperesthesia* is invariably present as a manifestation of the nervous disturbance. The patient can not endure the slightest touch, though *anesthesia* may soon follow and in some cases takes the place of the hyperesthesia. *Vertigo* is not uncommon and *delirium* usually appears early though, in serious cases, death may take place before this stage is reached. The *delirium* is inclined to assume the active form and the mania may be distressing while in other instances it may assume the toxic character of typhoid and end in coma. Signs of *motor irritation* will be recognized early and these will be defined as muscular tremors and contractions. The tonic spasm is generally so pronounced that the head is drawn back and opisthotonos may be extreme. This rigidity is liable to extend to all the body muscles and the patient is in reality in a state of extreme rigor. Later the contractions of the muscles of the extremities may create flexions so that the arms are bent upon the chest, the forearm upon the arm and the thumb upon the palm; the thigh is also flexed upon the abdomen and the leg upon the thigh. The opisthotonos may, in fatal cases, be associated with trismus and in younger patients convulsions may supplant the rigor of the tonic spasm. The *facial muscles* are often affected by the same spastic action or a paralysis may take place in the facial and eye muscles at the same time giving rise to strabismus, inequality of the pupils, diplopia and ptosis. *Photophobia* often exists as a result of retinal hyperemia and the pupillary changes are variable. *Conjunctivitis* is quite a common symptom, while keratitis or iridochoroiditis, of a purulent character, is a serious complication. *Ptosis* is nearly always present. *Deafness* is also to be feared when the auditory nerves are involved, and otorrhea is a common sequence of the disease. A *monoplegia* or a more serious paretic hemiplegia often attends or follows a severe or protracted run of meningitis.

The *temperature* is not usually as high as would be naturally expected in a disease so serious as this. The moderate fever which soon follows the onset is often lost sight of inasmuch as pain and the tendency to chilliness absorbs the attention of both patient and physician. The temperature may, however, rise as high as 104° or 106° F., and this often takes place just prior to death. In the average case when the temperature rapidly rises high it soon falls to 102° or 103° F., and remains at about this point, with occasional exacerbations, until defervescence takes place by lysis. The *pulse* keeps pace with the temperature, being slightly accentuated at first, but later it may become more feeble and frequent as the debility increases. The respiration varies according to the conditions, being often irregular or possibly depressed when there is pressure upon the respiratory center. In grave cases Cheyne-Stokes breathing may appear. "*Leukocytosis,* moderate or severe, is constant, the increase afflicting principally the polynuclear leukocytes."

The *cutaneous* symptoms are of unusual importance in the diagnosis of this disease. In the early period a peculiar pallor or lividity of the skin is often detected and this may also affect the exposed mucous membranes. *Herpes facialis* is also extremely common and in many instances it may invade the trunk and extremities as well. A *petechial eruption* is, however, more characteristic and inasmuch as it is quite general in its distribution, it has given rise to the term "spotted fever." It may appear as an erythematous blush in the beginning and in grave cases it may be associated with purpuric spots or even ecchymotic extravasations. Like the eruption in typhus, it does not disappear on pressure. In some cases it is limited to small areas but is often wanting, particularly in the sporadic cases. Other eruptions, urticaria, sudamina, rose-spots, pemphigus and ecthyma have been observed and gangrene may follow as the result of pressure or trophic insufficiency.

Arthritis, with joint deformity similar to the arthropathies in spinal cord diseases, is an occasional symptom. Renal disturbances are not generally in evidence, though the urine may often be albuminous and decreased in quantity, while glycosuria has been noted. Hematuria is often a feature of malignant cases.

Kernig's sign is now regarded as a valuable symptom in diagnosing the disease. It is illustrated by the inability to extend the leg when the patient is in the sitting position with the thigh flexed upon the abdomen and the leg partially flexed upon the thigh. This does not apply in the recumbent position unless we first flex the thigh upon the abdomen. The cause of this resistance to extension is explained by the extreme contraction of the flexor muscles. The same test is often applicable in other meningeal affections and the absence of this sign must not be accepted as a proof that meningitis does not exist. The *Babinski reflex* is often present and this test is made by irritation of the sole of the foot causing thereby the extension of the big toe. *Ankle clonus* is also a frequent sign of meningitis.

The *complications* which occur in this disease will include the ocular and aural inflammations with consequences already mentioned, but more particularly the pneumonic affection which is so closely allied. Pleurisy, endocarditis and pericarditis are likewise frequent sequelae, while hemorrhagic nephritis, chronic hydrocephalus and abscess of the brain are rare.

Anomalous Forms.—The *malignant form* of cerebro-spinal meningitis will be recognized by the severity of the chill, headache and coma and the fatal termination in sudden collapse. The fever is not marked, the temperature is often subnormal and there is a progressive slowing of the pulse until it falls to fifty or forty beats per minute. In these cases the eruption is absent or takes the form of purpura, the respiration is labored, and the urine is often scanty and albuminous. The *abortive form* is marked by the severity of the symptoms which usually subside in a day or two and are followed by a rapid convalescence. The mild cases are similar, only the cardinal symptoms are not severe, the fever is slight and the disease is not readily diagnosed except by the aid of a prevailing epidemic. The *intermittent form* is characterized by the remission or the apparent intermission of the typical symptoms every day or every second day, followed by an exacerbation of the fever. A *chronic form* is described when the characteristic symptoms are prolonged beyond the usual duration and prostration with febrile recurrences are marked features.

Diagnosis.—In making the diagnosis a physician is usually guided by the prevalence of an epidemic. The characteristic symptoms, however, particularly the retraction of the head, the tremor, muscular rigidity, eruption, delirium, headache and fever, will be recognized by an experienced observer. Among the special diagnostic features we must consider Kernig's sign of unusual value. Quincke's lumbar puncture furnishes the most positive diagnosis. The patient being placed upon the right side with the knees drawn up and the back bowed, a small aspirating needle is inserted about one-half an inch to the right of the median line between the third and fourth lumbar vertebrae. This should enter the spinal canal at a depth of two or three centimeters in children and four to six centimeters in the adult. The fluid may be collected, drop by drop, in a sterile test tube and subjected to microscopic and bacteriological examination. It may be necessary to repeat the puncture in case the test is negative. If cerebro-spinal meningitis exists, the diplococcus intracellularis will be found. Lumbar puncture will be unnecessary when the diplococcus is found in the nasal secretions. When tuberculosis is suspected a guinea pig may be inoculated for the sake of diagnosis. E. M. Squires has found the following sign invariably present as early as the third or fourth day in meningitis. It consists in rythmical dilatation and contraction of the pupil and is obtained by placing the child's head, face upward, between the knees of the physician, its body being supported. During gradual and forcible extension of the child's head on the spine produced by traction the pupils dilate, while they contract when the head is flexed on the chest.

The *differential* diagnosis will be required in many respects. *Pneumonia,* which may coexist or confuse, will be differentiated by the bacteriological examination and the symptoms as well. In the latter we shall find motor spasm and tremors, but as the meninges of the convexity is more frequently affected, there will be less retraction of the head.. The myalgic pains are also less pronounced and we may in addition note the fact that the pneumonic localization precedes the meningeal symptoms. *Tubercular meningitis* has its particular history, the prodromes anticipate the attack for a long time and the invasion is gradual. The abdominal retraction is more marked than in the cerebro-spinal form, while the retraction of the neck, hyperesthesia and other nervous signs are not so extreme. The eruptive symptoms are not usually present and the respiratory and pulse changes are characteristic. Tubercular meningitis is usually fatal and some other focus of the infection is generally found. *Typhoid fever,* in its cerebral form, may have many symptoms similar to meningitis, but its slower development, its peculiar temperature range, the rose-spots and the seroreaction, together with the absence of vomiting and muscular pains, will settle the diagnosis. *Typhus fever,* will be most difficult to differentiate. In this, however, the spots are more constant and numerous, while the rigidity and retraction of the neck are uncommon. The typhoid state is also more pronounced and its duration is more clearly defined.

Prognosis.—The prognosis in cerebro-spinal meningitis is always grave, the mortality varying from 20 to 80 per cent. In the mild forms it is possible for recovery to take place following a slow convalescence after the fever has subsided. Every day that the patient survives after the fifth day adds to his chances for recovery. The abortive form also may yield after a brief duration, but these are not so common as the text books would indicate. We find some cases, where traumatism has been a factor, which respond to remedies and hospital care. The severe cases, however, may be regarded as hopeless from the very first. Much depends upon the complications and the severity of the cardinal symptoms. Children furnish a higher percentage of mortality than adults, it being fatal in nearly every case under two years and somewhat less severe in those under five. Malignant cases generally terminate fatally in a few days or a week. A remission of symptoms must not always be regarded as a sign of improvement for a relapse usually succeeds.

Treatment.—Under this subject we must take into first consideration the *general management*. This, of course, will include proper isolation and, more than all, absolute quiet of surroundings. Nothing must be allowed to excite the patient and to this end it will be helpful to have the room darkened. The *diet* should be liquid, corresponding to that required in all febrile cases. All kinds of local treatment have been employed, from the application of blisters, cupping, leeching, Pacquelin's cautery, etc., to the hypodermatic injection of the perchlorid of mercury. The majority of these methods have been abandoned for the *continuous application of the ice-bag* to the head and spine. This evidently is bound to continue as our best means of relieving the terrible basilar and cervical pain. Whether it gives relief to the myalgic pains is somewhat questionable, but in all probability it does, as the ice is usually comforting to the patient and should be continued until there is positive evidence of permanent relief of pain or the temperature drops to normal. The cold bath has been advised, but this certainly is not agreeable to the patient, to say nothing of the shock to the already irritated nervous system. The relief of pain is the great desideratum and we have sufficient evidence that the application of ice is a great adjuvant in this respect. Quincke's *lumbar puncture,* which is a valuable measure for diagnosis, has been often relied upon to relieve pain as the withdrawal of spinal fluid will possibly lessen the pressure for a time. This procedure, however, rarely accomplishes much in this respect and laminectomy is required to establish a free drainage.

The *medical treatment* has so far been decidedly disappointing, except as it pertains to a clearly established symptomatology which is generally difficult to obtain when the patient is unconscious and suffering from excruciating pain. Thus *morphin,* in 1/4 grain or larger doses, combined always with a proper proportion of atropin, is probably our best agent for combating the excruciating pain. This is best administered hypodermatically and it will require repetition in severe cases at least once or twice daily until there is a marked relief of pain. *Opium* has been employed but it is not usually successful, though, in the form of deodorized opium or paregoric, it may be required when children are afflicted. *Sedatives* have been employed with the hope that a relief of tonic contractions will, in a sense, lessen the exacerbations of pain and particularly the myalgic involvement. Thus bromids and chloral have been used for a long time and in many instances antipyretics like antipyrin, acetanilid and phenacetin have been employed, but it is questionable if any of this class of remedies is expedient or safe. Among other remedies *quinin* has been used in specific doses but no appreciable results have been obtained by its use. ***Bichlorid of mercury*** has had its advocates who have used it from the beginning of the fever, in doses of 1-24 of a grain every three hours. ***Potassium iodid,*** in its usual dosage, has also been recommended, but we have no record of any satisfactory results from the use of either of these remedies. ***Mercurial inunctions*** have been tried with doubtful effects, but it is certain that *iodoform inunctions*—ten per cent—have given some relief, particularly with tuberculous complications. The *convalescence* will require a far different line of treatment, which may be decided upon according to symptomatic expressions and a wide experience as to the value of various tonics and other adjuvant treatment. The paralyses which attend this disease should be treated in accordance with suggestions already given under that subject. Lumbar puncture with aspiration of cerebro-spinal fluid and the injection of one per cent solution of lysol is a recent treatment which deserves trial. Again, Dr. Wolff, bacteriologist of the board of health of the city of Hartford, advocates the injection of diphtheria antitoxin in massive doses—10,000 units—early. This has given some good results in a recent epidemic in Connecticut and New York.

The *homeopathic remedies* will give relief when prescribed on a broad symptomatology. ***Aconite, belladonna*** and ***stramonium*** will often be suggested in the very early stages when the fever and the cerebral congestion give indications of their characteristic symptoms. ***Gelsemium*** seems to correspond to the symptoms of pain, rigidity and the head retraction better than any other remedy; it refers also to the myalgic pains in the extremities, the decided nervous irritation, the chill and fever, the profound prostration and the paralysis which affects either the limb or the ocular muscles. It may be given in the tincture or in potency, though my choice is the tincture, at least in the early stage, in five or ten drop doses at least four to six times daily. *Cimicifuga* is naturally allied with gelsemium as it has a wide sphere of action upon the cerebro-spinal system. It refers more particularly to cases with a neurotic predilection and the muscular cramps, with muscular soreness, will call attention to it, while we are also prone to think of it always when the patient is a female. The brain symptoms are always accentuated when this remedy is demanded and the throbbing sensation, with the feeling of outward pressure, is always present. When this remedy is demanded gastric disturbance will also be noticed as a result of the spinal irritation. *Bryonia* is always suggested when the exudation is first established and the characteristic stitching pain is a guiding symptom, though we must remember that, excepting mild cases, we rarely get any well defined expression from our patient. However, with the pathology in mind, it is evident that the pains of bryonia must be present and the remedy should be given when other remedies are not clearly suggested. *Cantharis* is similar to bryonia in its action but it is usually indicated in an earlier stage. *Veratrum viride* is also a remedy for the early stage, but it applies more particularly to a symptomatology in which the congestive symptoms are marked. Therefore we should expect it to be indicated when cardiac excitement, high tension pulse, high fever and even apoplectic symptoms are prominent; the pains of veratrum viride are invariably associated with convulsive twitchings. When the fever assumes the typhoid character we may expect a symptomatology which calls for *baptisia, echinacea, rhus tox, arnica* or *arsenicum.*

In the later stages we may have occasion to use several other remedies prominent among which are *helleborus niger,* which covers many of the cerebral symptoms in the disease and is certainly applicable when the effusion is extreme. In like manner we may refer to *apis mellifica* when the general edema is pronounced and the kidney secretion is impaired and with both of these we may always compare *apocynum,* and *stigmata maidis, digitalis, adonis vernalis, hydragogin and crategus* when the cardiac weakness is pronounced. *Cicuta virosa* is an unusually valuable remedy when opisthotonos is a characteristic symptom and it applies also to the typical delirium of the disease, while the spastic and tearing pains are also in evidence. *Strychnia* and *enanthe crocata* may be compared with cicuta. *Cocculus* will often be indicated when the least motion disturbs the brain; nausea will be a prominent symptom when this remedy is demanded. *Cuprum acet.* is a valuable remedy with children when abdominal cramps are a feature of the disease; it is also indicated when the stupor is extreme and even in the sleep the spasmodic twitchings continue. In line with these remedies we shall find indications for *hyoscyamus, cannabis indica, zinc phos., physostigma, ethusa* and *hydrocyanic acid.* When symptoms of collapse appear we shall naturally think of *camphor* and *veratrum alb.* in addition to the usual heart stimulants.

ERYSIPELAS.

Erysipelas, previously known as St. Anthony's fire, is an acute contagious disease characterized by a dermatitis, with all the signs of inflammation, and a peculiar tendency to spread. It is attended by a high fever of short duration and it may occur endemically or epidemically.

Etiology.—The disease is an ancient one, dating back to the time of Hippocrates. As early as 1840 the parasitic cause was maintained by Henle, but it remained for Koch, in 1880, to demonstrate the existence of the specific streptococcus in the lymph-vessels and the lymph-spaces of the skin. One year later Fehleisen succeeded in isolating, cultivating and inoculating with the erysipelococcus. Since this time the specific cause of erysipelas has been recognized in the *streptococcus erysipelatis* of Fehleisen. These may be defined as diplococci, arranged in pairs or more frequently as streptococcic chains found in the lymph-vessels and the cutaneous connective tissue in large colonies. The erysipelococcus has a close resemblance to the streptococcus pyogenes. The organism, acting as a local irritant, produces a dermatitis and from this focus a constitutional infection is established through the agency of a toxin. An *abrasion* of the skin or slight injury is usually necessary for the introduction of the organism, though in all probability skin diseases may favor its development to a certain extent.

It is, therefore, transferred by direct contact and is often rapidly spread by the surgeon when it occurs in surgical work and complete and thorough isolation is not established. Surroundings which are not hygienic are also favorable factors in its development. It may perhaps be transmitted by fomites and some hold to the theory of intravascular infection through the agency of the lungs and the digestive tract. If the former theory is true, it may be conveyed through the atmosphere. It frequently occurs, in lying-in women where inoculation is favored by the careless physician who has perhaps recently cared for a case of erysipelas.

Among the *predisposing causes* Anders places much stress upon the seasons. He claims that the greatest number of cases is liable to exist between the months of August and April, while between April and August there is a steady decrease in its epidemic occurrence. *Age* is also given as a possible factor, the greatest period of frequency being noticed between adolescence and early adult life. It is a question if this theory may be accurately applied. *Males* are said to be afflicted more than females, family predisposition has a slight influence and a previous attack may predispose. *Chronic diseases* like Bright's, alcoholism, phthisis and heart disease may favor the receptivity of the organism.

Pathology.—The erysipelatous inflammation in its uncomplicated form leaves little for post mortem observation except the inflammatory edema. The cocci are found mostly in the lymph-spaces and in the area of extending inflammation at the periphery. They are also found in the lymph-vessels of the surrounding healthy tissue, but here they are generally conquered by the phagocytic leukocytes. When the inflammation exends to the subcutaneous tissue suppuration usually follows. It is claimed by some that an acute meningitis or thrombosis of the lateral sinus may follow from its cerebral extension. The septic process may be established by infarcts in the lungs, spleen and kidneys and a general pyemic infection may be developed. Pericarditis and endocarditis may be sequelae.

Symptoms.—The most frequent form of erysipelas which the physician is called upon to treat is regarded as idiopathic inasmuch as the entrance of the organism can not always be traced to an infectious lesion, though this always exists. A *period of incubation,* varying from one to eight days, frequently exists and this is characterized by the prodromal symptoms of a slight infection. A chill usually announces the invasion, though this may often be wanting. The affected part will soon present the symptoms of local inflammation. A *circumscribed area* becomes red, swollen and indurated, with secondary symptoms of heat and pain. The head is usually the seat of the lesion, the bridge of the nose, the ear or the chin often being first implicated.

From this elevated inflammatory patch the swollen zone spreads upward over the forehead and laterally toward the ears until the whole of the face is involved. The edema distorts the physiognomy and often closes the eyes while the inflammatory process may then extend to the body and limbs. *Blebs* may form on the ears and the eyelids and minute vesicles are dispersed throughout the inflammatory area. A serum exudes but soon dries upon the skin, desquamation taking place as the dermatitis spreads to neighboring parts. *Suppuration* may follow and deep abscesses may develop when other pyogenic organisms are present. In the average case the local symptoms do not last much longer than four to six days. The mucous membrane may rarely be affected primarily or by a secondary extension from the skin. *Gangrene* may be associated with the deep seated varieties. The *local symptoms* will, of course, vary according to the variety of erysipelas which may prevail. In the *phlegmonous* or cellulo-cutaneous variety suppuration will be a leading symptom, while in the *relapsing* form the local symptoms will recur at different intervals, the seat of the primary lesions being usually affected. In *erysipelas neonatorum* the inflammation follows an infection of the stump of the cord and then spreads to the thighs and genitals.

The *general symptoms* will be manifested by the *fever* which follows the onset, the temperature rising quite rapidly to 104° or 105° F. This usually subsides as the local conditions improve or it may terminate suddenly by crisis on the fifth or seventh day, to be occasionally followed by another rise in case of inflammatory extension. The pulse is frequent, in proportion to the fever, but usually of good volume. In some instances the febrile action may assume a *typhoid character,* the coated tongue, the stupor and delirium simulating the toxemic features of that disease. The *urine* is often scanty and albuminous and nephritis may be a sequela either as a direct sequence or as an exacerbation of a preëxisting chronic nephritis. In either case hematuria may be a symptom. The *digestive tract* is generally disturbed in proportion to the severity of the disease, nausea, vomiting and constipation usually being present. The *larynx* may suffer from an inflammatory extension to the mucous membranes, and *nervous symptoms* due to the toxins may be manifested by severe headache and extreme restlessness.

The *complications* depend greatly upon the extent of the inflammatory invasion and the previous health of the patient. *Meningitis* has already been referred to, but this is probably of rare occurrence. *Edema of the glottis* may appear when the inflammation extends to the mucous membrane of the glottis. *Endocarditis* of a malignant character has occasionally existed, while pericarditis and myocarditis may rarely attend the fever. *Pulmonary* complications in the line of bronchitis, pneumonia and pleurisy are possibilities, while *nephritis,* hemorrhagic in character, and glycosuria may occasionally be observed. *Purpura* is also mentioned as a complication when the blood conditions are predisposing. *Septic* and *pyemic* conditions are the most serious complications which are liable to arise in the course of erysipelas. We must not fail to mention the frequent association of such infectious diseases as typhoid, diphtheria and scarlet fever. *Acute articular rheumatism* is also frequently associated with erysipelas, appearing usually at the first manifestation of the fever.

Among the *sequelae* of this disease may be enumerated the *loss of hair* and the *cicatricial new formations* which result from destruction of tissue by the suppurative process; hyperesthesia, anesthesia and atrophy of the skin may also be sequelae. Joint affections and symmetrical gangrene of the fingers may develop after erysipelas. Otitis media is a condition always to be feared after facial erysipelas. Some of the diseases already mentioned among the complications may properly be regarded also as sequelae.

Diagnosis.—While a confusion often exists in the diagnosis of this disease, it is not to be regarded as a difficult matter to determine its existence. The acuteness of the disease, the character and location of the eruption, as well as its rapid spread, should be guiding points. The constitutional disturbance which always attends the fever should distinguish it from similar affections. *Erythema* has a more superficial redness and is not accompanied by local heat and swelling or constitutional symptoms. *Urticaria* appears in crops, itches intensely, has a short course and is characterized by pale-red circular wheals. *Eczema* is more localized, progresses slower, itches decidedly and has less swelling. *Eczema nodosum* is recognized by the nodosities in the vicinity of the joints.

Prognosis.—The outcome in this disease is not to be feared unless it is of a malignant character. If no complication exists, it will pursue a rapid course and end by apparent crisis. Any associate organic disease will naturally predispose unfavorably. Age and general debility mitigate against a prompt recovery. It is claimed that erysipelas exerts a favorable influence on some chronic diseases and malignant growths and for this reason the inoculation process is employed in the treatment of cancer.

Treatment.—Isolation is a proper precaution to carry out in the treatment of these cases and the most painstaking care should be exercised by the attending physician not to disseminate the infection. The patient should remain in bed until the fever and the local inflammation have disappeared. The diet should be nourishing, easily digested and liquid or semisolid. Local treatment is rational inasmuch as we have to deal with a localized infection of the skin and subcutaneous tissue. Carbolized vaselin, zinc oxid and calendula cerate, wet dressings of boric acid, bichlorid (1 to 5,000) and ichthyol certainly do allay the burning and itching and limit the spread of the infection. Cool water alone often relieves the burning sensation. The old cranberry poultice—unpleasant as that word sounds in modern practice—has often been serviceable. Dusting the parts with powdered oxid of zinc, or the subnitrate of bismuth may be efficacious. Ichthyol is recommended by many and it may be used with glycerin or collodion (2 drams to the ounce). Curtis advised the use of sodium sulphate applied over a piece of gauze protecting the parts and to this is added at intervals a sprinkling of cold distilled water. Formic-iodid is another good preparation. The tincture of iodin may be painted around the inflamed parts to prevent the spreading. Heuter's plan of injecting hypodermatically a 2 per cent solution of carbolic acid or a 1-4000 corrosive sublimate, just beyond the edge of the advancing dermatitis, is worthy of trial.

Many cases are so mild as to require no local treatment. *Prophylaxis* will include the care of the skin which is not affected in order to prevent reinfection or autoinfection. Besides the general bath daily the healthy skin may be washed at more frequent intervals with a 4 per cent boric acid solution. A frequent change of linen will also add to the cleanliness and comfort of the patient.

The *constitutional* treatment will also need some attention and in case the pulse is high and the heart erratic, strychnia or other stimulants may be required. The high temperature will call for febrile remedies such as *aconite, belladonna, bryonia, baptisia,* etc., the same as in other fevers. In the later stages should anemic conditions attend we may need an entirely different class of remedies. *Pain* may be such a factor that opiates may be required unless the indicated remedy is sufficient to correct the disturbance. In like manner the *nervous symptoms* may require special attention, and, should insomnia, nervous irritability or delirium attend, we may need *hyoscyamus, hydrobromate of hyoscin, stramonium* or even *chloral* to obtain temporary relief. The complications must be cared for in accordance with suggestions named under other subjects.

The *homeopathic remedies* are of best service in this disease and usually they meet all the requirements. *Belladonna* is probably first suggested by the erythematous blush, the congested face, the carotid throbbing and the cerebral manifestations. *Rhus tox.* is always suggested when the eruption assumes a vesicular form and the inflammation partakes of the phlegmonous character; in such instances the patient is usually restless. *Arsenicum* covers a multitude of cases when the typical exhaustion, burning of the parts and excoriating tendency exists. This remedy will apply with much force to the chronic cases or when the typhoid symptoms ensue. It is also to be employed when there is a peculiar migrating tendency of the infection. *Apis mellifica* applies to the edematous condition which is not confined alone to the face. In such cases the characteristic stinging sensation is usually present and the urinary excretion is diminished. *Cantharis* will be useful when the eruption assumes the form of blisters, and should the suppurative process ensue we should naturally think of *tartar emetic, hepar sulphur, calcarea fluor., mercurius* and *silicea*. *Sulphur* also will have its sphere of action when there is some latent condition which acts as a disturbing factor and the totality of symptoms is not well marked. *Lachesis* is indicated in patients beyond middle life when prostration and depression are complaints and the skin has a dark or bluish appearance. *Zincum* seems to apply to those cases in which the erysipelatous inflammation affects the head and face alone and brain symptoms rapidly appear.

Echinacea is a remedy which has been excessively used by the eclectic school, who have employed it in heroic doses of the tincture. In conjunction with this they have used the red *sulphuret of arsenic,* in 1/50 grain doses, three or four times daily. Echinacea seems to apply when the septic conditions are quite general and there is a marked tendency toward boils or malignant ulceration. It may be compared otherwise with *arnica, baptisia* and *rhus tox. Dulcamara* has a sphere of action in which there is an activity of mucous membranes as may be evinced by the symptoms of coryza. It is probably efficacious when the mucous membranes are affected. *Veratrum viride, borax, hydrastis, croton tig., ruta graveolens* and *kali carb.* are often intercurrent remedies.

SEPTICEMIA.

The term septicemia is used to define a toxic condition arising from the invasion of the blood and tissues by pathogenic bacteria with or without any visible site of infection. It should be distinguished from sapremia which refers to the entrance into the blood of toxins produced by putrefactive bacteria. Septic infection is an expression which relates to the direct absorption of poisons from a suppurating focus.

Etiology.—Septicemia has a bacteriological cause either in the existence of micrococci, streptococci or staphylococci. Despite the various opinions which have previously been maintained we must admit that the condition exists when either or all of these organisms are at fault.. More than this, the clinical signs are apparent when a septic intoxication follows from the absorption of the poisons which they liberate. The infection is usually introduced through the agency of a wound which is intentional or accidental. The uterus also furnishes an atrium for microbic invasion in the case of miscarriage or operation, while a septic infection may arise from an infection which enters the system by a slight abrasion or a superficial wound. The mucous membranes often act as agents for the absorption of a virus from their surfaces as may be instanced by the entrance of gonorrheal, typhoid or dysenteric organisms directly into the system. Inflammatory rheumatism and tonsillitis also furnish examples of this method of infection. Intestinal toxemia is another factor in systemic poisoning, the micro-organisms acting either directly as the infectious agents or more commonly through a sapremic poisoning by means of ptomaine development. Septicemia may also arise from a bacterial infection or a sapremic poison when deeper tissues suffer from a local infection, as may be illustrated in the case of osteomyelitis.

Pathology.—The pathological changes, although not pronounced, will be recognized by the brownish color of the muscles, the ecchymotic spots in the pia mater and the dark appearance of the blood which is diminished in its coagulability, and reveals, by the microscope, the presence of micrococci and bacilli. The spleen is slightly softened and ecchymoses will be detected on the serous membranes of the pericardium and peritoneum. When septicemia is prolonged endocarditis and gastro-intestinal catarrh, with slight hemorrhagic extravasations, may be recognized. The lymphatics are also enlarged, while the liver and uriniferous tubules will be subject to the same form of catarrhal inflammation and edema. The internal organs will also present numerous inflammatory foci in some of which the necrotic process has already started. Bacilli are also found in these and other parts.

Symptoms.—*Sapremia* will present a symptomatology which is dependent upon either a local infection or putrefactive changes. An example of the latter will be found in "intestinal sepsis" caused by tyrotoxicon and possibly again where symptoms arise from the inhalation of foul odors. A chill of slight degree may or may not mark the invasion, while the symptoms of nausea, vomiting, diarrhea and colicky pains soon ensue. The temperature soon reaches 101° to 103° F. and may suffer occasional exacerbations. Prostration and secondary anemia usually follow and blood examinations will reveal a leukocytosis, a red cell reduction and a diminution in hemoglobin. *True septicemia* is characterized by an incubation period of four to six days and the onset is gradual though often announced by a distinctive chill. At first the temperature may rise rapidly and in severe cases it is liable to sudden hyperpyrexia, but the more common type is the continued form marked by morning remissions which may even be subnormal. The pulse is rapid at first and in the later stages manifests considerable weakness as cardiac failure ensues. Endocarditis may be a factor in some instances, though there is no appreciable murmur unless other complications favor it. A mild leukocytosis is generally observed and the micrococci may be found in the blood. Gastro-intestinal symptoms are represented by the vomiting which often prevails in the early stage and the septic diarrhea which follows later. The cutaneous features will be recognized by the punctiform hemorrhages into the skin and these may become so extensive that they form ecchymoses. Eruptions simulating that of scarlet fever, herpes and roseola may appear and jaundice is an occasional symptom. A condition known as septic nephritis sometimes exists and this is determined by the presence of albumin, epithelium, casts and blood corpuscles.

Diagnosis.—Sapremia and septicemia will be differentiated by the character of the symptoms already defined and by the discovery of the cause. In the former the early appearance of the symptoms and their prompt relief when the exciting cause is removed will constitute a marked feature for distinction. In the latter case the signs of the incubation period, the continued fever, the mental symptoms, the splenic enlargement and the nephritic complications will be of great service in making a diagnosis. The blood examination will furnish the most positive evidence.

Prognosis.—So much depends upon the extent of the infection, the general health of the patient and the scientific management of the case that a prognosis must be given with some degree of latitude. In sapremia the patient may be out of danger in twenty-four hours or a fatal termination may come in this time, the infection being so virulent. In septicemia the progressive tendency is noticed in regard to all the symptoms and thus a fatal end may come after a prolonged illness or by proper treatment a recovery may gradually take place.

Treatment.—The first object of the physician will be to *remove the cause* and this will often require the aid of the surgeon. Next, we must *support the patient,* who is suffering from a slow process of poisoning. This will be accomplished by means of a diet which should consist mainly of nourishing foods like eggs, milk and rich broths, being, of course, varied according to the temperature range and the physical condition of the patient. *Stimulants* will often be required and for this purpose it may be necessary to resort to alcohol and strychnia. Remedies administered according to their antiseptic value have a sphere of usefulness which will, no doubt, be better understood in the future. The *anti-streptococcus serum* is strongly recommended by many and, no doubt, is a valuable agent in many cases. Twenty to thirty c.c. of this may be injected every six to eight hours until a decided improvement is apparent. The normal salt solution has, no doubt, been our most useful agent for tiding over the stage of depression following the prolonged toxemia. The development of modern aseptic technique which now exists in the practice of surgery and obstetrics is, however, the most important feature of treatment, for this precludes the septicemia itself. The internal remedies will be mentioned under the subject of pyemia.

PYEMIA.

This is a febrile disease due to sepsis from the metastasis and absorption of pyogenic organisms.

Etiology.—The *bacteriological* cause of the disease may be traced to the staphylococcus and the streptococcus, though other specific organisms may be included. The infection is introduced through the blood vessels, the bacteria producing a thrombo-phlebitis or, in rare instances, a thrombo-arteritis. From these points emboli may be disseminated throughout the system; these, together with the bacteria which circulate independently in the blood, give rise to new septic foci. The lymphatic system is also a carrier of pyogenic bacteria, but this is not a frequent medium for their spread since they offer much opposition to the invading host. The pyogenic embolus may also start from within when for instance the condition of ulcerative endocarditis exists or when the appendix is the seat of micrococci infection. Among predisposing causes may be mentioned epidemics, the more frequent affliction of males, the prevailing influence of spring and traumatism.

Pathology.—*Thrombosis* of the veins in the region of any tissue and the secondary changes which follow are usually the initial pathological features. This may take place in the pulmonary veins or it may be present in the liver, kidneys, brain and other structures. Both thrombi and emboli may establish metastatic abscesses in the lungs, liver, spleen, kidneys and other internal organs. The small abscesses formed by the original septic focus may communicate or unite with other foci and thus establish large abscesses. *Infarction* may also be the primary cause of a septic dissemination. The *skin* will also present hemorrhagic extravasations and pustular eruptions, the mucous membrane of the stomach and large intestine may be invaded by ulcerations, favoring suppuration and the serous membranes will often be the seat of purulent inflammations and hemorrhagic extravasations. The *muscles,* the submucous, subcutaneous and osseous tissues may be the seat of abscess development while ulcerative endocarditis and suppurative myocardial degeneration are common pathologic lesions.

Symptoms.—A short period of *incubation* always characterizes the suppurative development and the onset is generally announced by a chill. A low grade fever may precede this for some time, while in some instances there may be no suggestion of a chill. The character of the chill will vary decidedly and it is inclined to reappear at intervals. The *fever* is marked by the rapid rising temperature which generally succeeds the chill; it has a more particular peculiarity in its intermittent or remittent character. At times it may reach the normal or even the subnormal point and remain there for a short period. *Sweating* always succeeds the drop in temperature and subsequent to this the prostration is pronounced and persistent. The *pulse,* accelerated at first, assumes a rapid and feeble character as the absorption continues and the heart is depressed. The *respiration* will be labored when sepsis is pronounced, while dyspnea, cough and purulent expectoration will be recognized in case the lungs are the seat of septic foci. *Pain* will be a complaint when large nerves are implicated by the suppurative process or the abscess is superficially located. *Cutaneous symptoms,* aside from the sweating, will be characterized by an occasional jaundice, particularly if the liver is involved, and subsequent shrinkage. Eruptions in the form of erythema, purpura and pustules are common, while there is often a general hyperesthesia. Hepatic and splenic abscesses will be diagnosed by the symptoms and physical findings which point to the direct involvement of these organs. The mental condition will be characterized by the delirium which attends the fever and the coma which appears in the final stage of a fatal case. When the meninges are afflicted with a purulent exudate, hemiplegia, ocular palsies and aural disturbances are occasional sequelae. *Joint swelling* sometimes follows from metastasis while invasion of the bone may give rise to the symptoms of acute osteomyelitis. *Albuminuria* is sometimes an attending symptom, while the microscope may reveal the presence of casts, micrococci, pus and blood corpuscles. The *blood changes* are characterized by a leukocytosis, a slight reduction of red corpuscles and a moderate structural change. A *septic* condition may be recognized by the general intoxication aside from the chief symptoms of pyemia.

An *abscess* will be determined by the local signs of tenderness, pain, swelling and fluctuation. These are often difficult to find. When the lungs are affected we must be guided by the physical findings, the expectoration and the respiratory disturbance. Abscess of the liver gives the additional evidence of jaundice; when the kidneys are affected the albuminuria, pus, and blood will be guiding symptoms, while a diarrhea is suggestive of intestinal involvement. Superficial abscesses are easy to determine and the microscope will reveal the pyogenic bacteria.

Diagnosis.—The symptoms which characterize pyemia must be differentiated first from those which are typical of septicemia. In the latter suppuration is not so distinct a factor, the chill only marks the onset, the temperature is of the continued type, sweats are not a part of the symptomatology, prostration and emaciation are less pronounced, jaundice is not usually present and metastatic abscesses do not exist. *Malaria* will be differentiated by a more characteristic intermittent fever, by the specific action of quinin and the presence in the blood of the malarial plasmodium. *Acute miliary tuberculosis* and *typhoid fever* should offer no confusion when such cases are properly studied. When *malignant endocarditis* is present the cardiac murmur will attend the peculiar febrile manifestations, the sweating and the prostration.

Prognosis.—The prognosis may be regarded as unfavorable. The removal of the suppurating cause is vital to recovery and this depends upon our surgical help and the aseptic care of the suppurating lesion. If the pyogenic process can not be overcome, we should expect only palliation from our treatment.

Treatment.—The treatment of this disease should be primarily surgical. Indicated remedies will be of slight service before the offending foci are removed. After this, however, much will be demanded of the physician. *Supporting tonics,* stimulants and a nourishing diet will be of service according to the requirements of the case, which any experienced physician should understand. The *internal remedies* which are naturally suggested both for pyemia and septicemia should be administered according to clearly defined symptoms. At best we can only mention some which are of constant use in pyemic and septic conditions. The *internal antiseptics* are naturally thought of first and this will include those previously mentioned —beta naphthol, benzosol, salol and methylene blue. The latter at present is my favorite, though I doubt if internal antiseptics will always give satisfaction in the treatment of this disease. *Strychnia* and *digitalis* may be required when the heart needs artificial support, and *alcohol* is, no doubt, of service in adynamic conditions. Among the homeopathic remedies we first think of *arsenicum,* which certainly is almost the similimum in this disease. Allied with this is *cinchona* which, next to arsenicum, has more symptoms similar to the pyemic condition than any other remedy. *Arnica* goes back to the traumatic infection and covers many of the symptoms which attend or succeed the tissue destruction. Remedies which apply to the organs which are affected by the embolic or thrombotic invasion should also receive thorough consideration. Febrile remedies like *belladonna, baptisia, echinacea, rhus tox, eupatorium* and *cedron* must always be employed according to symptomatic demands. *Hepar sulphur, silicea, calcarea, ferrum, mercurius* and *lachesis* will have their sphere of usefulness. *Eucalyptus* has been highly recommended by Dr. Cowperthwaite for its antiseptic properties.

RHEUMATIC FEVER.

The term rheumatism has been subject to so much misuse that it is well to call particular attention to the definition. It is an acute infectious disease characterized by a peculiar febrile action, the acute swelling of one or more large joints with a possible secondary involvement of the endocardium, pericardium, myocardium and other tissues.

Etiology.—Numerous theories have prevailed regarding the cause of this disease and even yet no positive evidence as to the real micro-organism which produces it has been secured. It has taken a long time to overcome the prevailing idea of a generation that *uric acid* is the causative factor and even now this theory is maintained by many in the profession and a majority of the laity. The *metabolic theory* as first illustrated by the claim of Prout that lactic acid, developing in the system from a faulty elimination, or by Latham's modification that this, together with other substances, acted as the irritating cause, have also been abandoned as untenable theories. In like manner the suggestion of Mitchell that the local manifestations are trophic or the result of an imperfect metabolism following a nerve lesion are not now accepted. The etiology is not settled except the fact that it is an infectious disease of bacterial origin. The weight of early evidence pointed toward the staphylococcus as the probable cause. Netter isolated the streptococcus. Singer, in the examination of a large number of cases, discovered both the streptococcus and the staphylococcus and Achalme described a bacillus resembling that of anthrax. Poynton and Shaw demonstrated by a series of experiments on rabbits and monkeys that the diplococcus rheumaticus was the real factor which produced rheumatic polyarthritis and this is the view which now is most universally accepted. It is no longer held that the staphylococcus is able to produce the disease and, therefore, it is not considered as an affection similar to pyemic involvement. Beattie, of Edinburg, isolated an organism "corresponding in every detail to the diplococcus rheumaticus of Poynton" with the additional evidence, sustained by experiments on rabbits, that this micro-organism created the cardiac symptoms of rheumatic fever. Shaw, of Oxford, has more recently demonstrated that the micrococcus rheumaticus, the diplococcus rheumaticus and the streptococcus of chorea "are identical organisms and are the actual infective and causual agents of acute rheumatism." It has also been claimed that the tonsils are the atrium of the infectious invasion and the rheumatic fever has been considered as a disease which is secondary to tonsillitis. Many other diseases, particularly scarlet fever and gonorrheal synovitis, so closely simulate the joint affections of rheumatic fever that it is often difficult to make a clear differentiation.

Predisposing causes seem to be essential factors in the development of the disease and chief among these we may mention the exposure to colds, sudden changes in the temperature and the prevalence of an infective lesion like a septic wound or tonsillitis. The *seasons* are perhaps predisposing influences as a majority of cases usually occur during the early months of the spring, and *climate* is no doubt a factor inasmuch as the temperate zones contribute the most cases. *Occupation* is also a factor as those who are exposed to the cold and wet weather are frequently afflicted. *Age* seems to influence its invasion, for the young adults suffer most frequently and severely. *Sex* evidently is not concerned for an experienced practician will find it common among both men and women, though perhaps the former are more subject to it by their life of exposure. Lowered vitality, arising from fatigue, overwork and malnutrition, is invariably a favorable condition for the infectious acceptance and prolonged illness of any kind probably makes the patient more susceptible. *Chronic endocarditis* is closely associated with its development and *chorea* is claimed as a predisposing disease. The disease may be endemic or prevail in epidemics, one attack having a tendency to favor a recurrence.

Pathology.—Aside from the febrile sign of the infection we naturally look for the most distinct pathologic factors in the joint enlargements, though in many instances these changes may be extremely slight. The synovial membranes of the affected joints are hyperemic and swollen. The serous effusion is sometimes marked, is turbid in character and contains fibrin, leukocytes and rarely pus. The periarticular structures may be infiltrated by this exudate, the tendinous sheaths may be somewhat inflamed and in extreme cases slight cartilaginous erosion may be found. The fibrin of the blood is invariably increased, while endocarditis, pericarditis or myocarditis are often a part of the pathologic findings.

Symptoms.—The *onset* is rather sudden, though a prodromal state, characterized by slight malaise, may precede this for a day or two. Some cases develop with a marked chill or at least chilliness, while others manifest no sign of this kind.

Joint involvement is the first symptom of distress, and this will be manifested by the severe pain which attacks the larger joints, like the knee, ankle, wrist, shoulder, elbow and occasionally the hips. The inflammatory invasion shows a decided tendency toward the migratory character or alternate affliction of one joint and then another. In like manner the pain is liable to manifest exacerbations and these again may be supplanted by periods of apparent freedom from suffering. The pain is rarely limited to one joint and on account of this polyarthritis the patient can endure only the slightest movement without extreme suffering. The affected parts display a decided redness in the earliest stages, there is tenderness to the touch and swelling soon ensues. The joint enlargement is mostly due to the synovial effusion, but the inflammatory edema may affect also the periarticular structures, even the muscles partaking of the swelling. In mild cases these local symptoms affect only a few joints and one of these often presents the characteristic symptoms. Kohler claims that the disease may exist without the slightest joint implication. In severe cases, all the important articulations may be involved, including the phalangeal, the symphyses of the jaw, ribs and the sterno-clavicular attachments. Ankylosis is a rare sequence, though more extensive local changes may take place in case of suppuration following a mixed infection.

The *fever* is characterized by a moderate temperature (102°-104° F.) appearing in the early stages coincident with the joint involvement. This may, in rare instances, be marked by the development of hyperpyrexia, in which the temperature may rise as high as 106°-108° F., death ensuing unless there is a prompt remission. In these extreme cases delirium is a serious attending symptom. The average case displays a peculiar tendency to slight exacerbations and remissions of temperature by profuse sweats. The *pulse* is rapid, though usually soft and full unless cardiac or septic complications intervene. In such cases it will manifest much weakness and irregularity, the same often arising when the nervous irritation is extreme. The blood count will show a reduction in red cells and a leukocytosis will be evidenced proportionate to the severity and prolongation of the disease. The *cardiac involvement* is always a feature of anxiety in this disease. Acute endocarditis, usually of the simple form, is the most frequent attending condition and this will be recognized by an apical systolic murmur showing a pathologic involvement of the mitral valves. In case there is a further cardiac complication of long standing, dyspnea, palpitation and other signs of heart weakness will be present. Pericarditis, with an effusion, varying in character and extent, or myocarditis, representing different degrees of mural weakness, may also complicate the endocarditis or exist alone and these will be recognized by their characteristic symptoms.

The *skin* furnishes a positive objective symptom in the free perspiration which usually is constant, though a copious sweat occurs when there is a remission in the temperature. The odor is "sour" and the reaction is acid, though it may become neutral or alkaline later. Sudamina are frequently present in crops, discolorations are often noticed, while urticaria, erythema and purpuric patches, which may be attended by sloughing, are often present. In addition to these, cutaneous ecchymoses and mucous membrane hemorrhages are occasional symptoms. The *nervous symptoms* are often prominent, restlessness, insomnia and active delirium attending the periods of temperature exacerbation. In case of hyperpyrexia these may attain the degree of active delirium. The urine is scanty, acid in reaction, of high specific gravity, with an abundant deposit of urates. Albumin may be found during the early febrile action and the chlorids are diminished.

The *complications* in rheumatic fever are serious, and have already been referred to as a part of the symptomatology. The *hyperpyrexia* is exceedingly dangerous when, by the persistent delirium, it indicates a cerebral involvement. It appears most frequently during the second week and is most common in first attacks. During this high fever the pulse becomes decidedly weak and erratic, the prostration is extreme and a prolonged stupor will indicate a speedy death. The *endocarditis,* described as a common feature of the symptomatology, is liable to become a serious condition in repeated attacks, in young patients, and when engrafted upon an old cardiac lesion. In serious and prolonged cases the secondary sclerotic changes and retraction of the segment may lead to an incurable affection of the heart. *Pericarditis,* independent of or associated with endocarditis, may assume the simple fibrinous or sero-fibrinous form, or in children it may take on the purulent character.

Myocarditis, though not so common, is characterized by a granular or fatty degeneration which leads to mural weakness and secondary dilatation. The cardiac complications are far more common in children than in adults, affections of this kind frequently supplanting, as well as following, the arthritic symptoms. The skin eruptions and the attacks of tonsillitis are also more common attending symptoms when children are afflicted.

Pleurisy is often a sequela or attending complication as the result of inflammatory extension from the pericardium. *Bronchitis* and broncho-pneumonia may be encouraged by the febrile state of inflammatory rheumatism, while *lobar pneumonia* is an exceptional complication. Pulmonary congestion is a natural and often a serious condition following the cardiac impairment. *Nervous complications* may be attained when the ordinary nervous symptoms reach a state of extreme delirium —particularly in the alcoholics—or when coma presages a fatal termination. *Convulsions* are not uncommon in extreme cases, mental perversions may follow a prolonged attack and meningitis is a rare sequence. *Chorea,* in children, is clearly associated with this disease. *Rheumatic nodules* are sometimes found attached to the tendons and fasciae underneath the skin. They are most numerous on the fingers, hands and wrists, though they may be found about the elbows, knees and the spines of the vertebrae. They are about the size of a pea and affect children more than adults, appearing usually during the febrile decline or when the cardiac symptoms are pronounced. These little deformities are firm, movable and painless, being composed of round and spindle shaped cells, without any associate inflammatory evidence.

Diagnosis.—The symptoms of sudden fever, polyarthritis, copious acid perspiration with the peculiar remissions of temperature, the cardiac murmurs and the attendant chorea and tonsillitis in children are guiding symptoms which should call attention to this disease. In the differentiations we must first take into consideration the *subacute rheumatism* which may follow or occasionally supplant the true type. In this the fever is mild and the arthritis is less intense, though, in children particularly, it is invariably associated with endocarditis or pericarditis. This form has also a greater tendency to chronicity. *Septic arthritis* will often simulate rheumatic fever, but the general pyemic state, the irregularly intermitting fever which precedes the joint involvement, the rigors and marked temperature exacerbations, the local suppurative signs, the skin discoloration and the more persistent symptoms will usually establish the differentiation. *Gout* will be recognized as a more chronic disease with only occasionally acute arthritis, the smaller joints are involved, the periarticular deformity is more pronounced and permanent, the affection may be confined to one joint particularly in the big toe. *Arthritis deformans* has none of the febrile characteristics nor any of the local signs of inflammation. It is a joint deformity, symmetrical in its involvement, which is characterized by the destruction of the articular structures and the osseous periarticular formations. A neurotic cause is generally found and a pseudo-ankylosis is an invariable sequence. *Gonorrheal arthritis* follows closely upon the urethral infection, a single joint, like the knee, or sternoclavicular, is usually affected, the arthritis is more pronounced than the signs of general infection, it is liable to pursue a longer course and gonococci may be found in the effusion. *Acute osteomyelitis* also affects a single joint in the beginning, the local symptoms are more intense, the epiphyses, and not the joints, are affected and the constitutional symptoms are more extreme. The *acute arthritis of infants* is generally confined to the hip or knee joint, the inflammation is purulent and general pyemic symptoms are in evidence. *Hemorrhagic joint diseases* must be differentiated by the character of the effusion and the other characteristic symptoms of scurvy, purpura, etc.

Prognosis.—The course of this disease will vary in different individuals, but the result is dependent upon the prompt and scientific treatment. Aside from the cardiac complications we need have little fear as to the result if the patient yields to the necessity of remaining in bed until every vestige of the disease has disappeared. The cardiac complications are always a source of much anxiety, particularly in children, but if these are carefully and persistently treated, there will be no fear of serious results. The subacute condition is liable to be a tedious prolongation of the disease.

Treatment.—The treatment of "rheumatism" has undergone various changes during the last few years, when it was considered as a joint disease per se. All kinds of local applications were resorted to for the relief of local pain, the object being to eradicate the disease in this way. Then the uric acid theory of Haig had its day and elimination was the principal object to be obtained, and, of course, this was not an unfortunate line of treatment. Inasmuch as the disease is now known to be infectious in character, a very different principle of treatment prevails. It is safe to say that in present day practice we may regard this disease as within the control of internal medication. This statement, however, does not properly include all of those joint affections and general pains which are erroneously included under the term "rheumatism." As a part of the treatment we must consider the necessity of placing our patient into bed and employing all the methods of care which apply to any acute constitutional disease.

Local applications have a certain degree of usefulness. In a majority of instances the joint pains will be relieved by a simple covering with cotton and a roller bandage. This will give more relief if the whole limb is encased, thereby maintaining an even temperature. In some cases a wet boracic acid dressing, properly protected by oiled silk and a bandage, will give relief to the excruciating pain. *Oil of gaultheria* is at present a popular local application and is used in the proportion of 1 part of oil of gaultheria to 2 parts of olive oil. It is well to saturate flannels in this and apply them firmly to the inflamed parts, covering with oiled silk or gutta percha. At present this, in addition to the dry cotton covering, is considered to be all that is required in the local treatment of the average case of rheumatic fever. In exceptional cases it may be necessary, on account of the extreme pain, to employ hot fomentations of various kinds. Flannels wrung out in hot alcohol water or some of the antiphlogistic dressings may palliate the severe pain for a time. Cold compresses and the ice-bag have been suggested by some, but these are at best unpleasant. The permanent relief, or at least the cure, must come from the internal medication, while local rest for the affected parts must always be secured.

The principle of *internal treatment* has undergone various changes, the main object being to overcome the specific cause. When the uric acid theory was accepted the *alkaline treatment* was thought to be of greatest consequence. For this purpose the alkaline salts, potassium citrate, potassium acetate and sodium carbonate, were used most exclusively. Ten to twenty grains of either of these, according to the exigencies of the case, should be administered every two or three hours until relief is obtained, and then at longer intervals. This plan of treatment is often applicable as an adjuvant measure in present practice. The free use of *alkaline waters,* however, has taken their place to a great extent. The object of *free elimination* is of importance, whether uric acid excess is or is not a factor in the disease.

The use of *salicylates* is, at the present date, the best of all the stereotyped forms of treatment. In my service in Cook County and Hahnemann hospitals I have had a good opportunity to watch the results of this form of treatment. The *salicylate of sodium* is the remedy most commonly used and 10 to 15 grains, repeated at first every two or three hours, and later given four to five hours apart, constitutes the dose for the average case in adults. If this remedy is given in the early stages and continued as suggested above, the most severe cases usually are relieved from the excruciating pain and the fever in from one to three weeks. Remedies indicated according to their symptomatology are then the only required agents. Not rarely, however, the salicylate of sodium is not well borne by the stomach and it will then be wise to change to the salicylate of strontium, which may be given in larger doses. After the acute exacerbations have subsided, *aspirin,* in 10 to 15 grain doses, may be substituted. In many obstinate cases it is well to add to the maximum dose of this 10 to 15 grains of potassium acetate. In a few extreme cases, for some unknown reason, the salicylates are neither endured nor do they seem to answer the purpose in treatment. In such instances *colchicin,* in 1/100 grain doses, invariably gives relief when it is administered every three or four hours. When there is marked relief it is best to discontinue this remedy for milder ones which may be suggested by the leading symptoms. The *oil of wintergreen,* in 10 to 20 minims every two or three hours, is often successfully substituted for the sodium salicylate. *Salicin,* in 10 to 15 grain doses, may be temporarily employed every two hours. *Salophen,* in 10 to 15 grain doses, in adults every four hours, is also considered as a good substitute for the regular salicylate. In the employment of any of these remedies it is advisable to alternate or combine with the alkaline treatment. When this method of treatment is carefully borne it is claimed that the cardiac complications are not so severe and I can say, from my observation, that this is true. Much care must be directed toward the constant danger of cardiac depression, and I am not so sure that the same degree of success will be attained by this treatment in private practice that has been observed in hospital experience. More than this, we must always remember that some patients will not endure any form of the salicylates and that a carefully selected remedy will work a safer if not so prompt a cure. *Potassium iodid* has had its advocates in the treatment of this disease, and *lactophenin* has also received some considerable praise. The use of *anti-streptococcic serum*, though suggested as a positive specific cure, has not as yet revealed a sufficiency of satisfactory results.

The *complications* which are liable to occur in any case of rheumatic fever must be carefully anticipated and cared for. The cardiac perversions are most frequent and dangerous. These will usually be corrected by the appropriate treatment described in different chapters, but as a general rule the remedies which overcome the infection will protect the heart. When cardiac weakness is indicated by a feeble first sound or the signs of general asthenia, it is safe to assume that the salicylates, if used, are depressing the cardiac action. If this condition arises from a previous cardiac disorder, it may be necessary to use the indicated cardiac stimulants. Pericardial effusion may demand relief by paracentesis. Another danger to the heart may exist during the period of convalescence when the patient is possibly allowed too much freedom. It is, therefore, a wise procedure to keep every patient in bed and under close observation until there is positive evidence of a perfect cure and a normal cardiac action. As the patient leaves his bed it may be necessary to resort to *massage* for the relief of joint and muscular stiffness, while outdoor life and tonic treatment may be required to re-establish the normal health. Indicated remedies may be needed for some time and a general warning should be administered against any excessive exertion which might injure the heart.

The *homeopathic* remedies are essential in the treatment of this disease and they may be successfully employed in many cases independent of the specific treatment. Before the salicylate of sodium was found to be of such value in this disease, I am positive that I have attained good results with the homeopathic remedies alone. I doubt, however, if the disease was cured so promptly and if the sequelae were less severe. Our aim in practice should always be to seek the truth and if I were to express my chief reason for the use of salicylates, I should claim for this practice a more prompt relief of acute symptoms and the lessened liability to endocarditis and other complications. If we abbreviate the stage of febrile action we are less liable to meet with cardiac sequelae. Among the important homeopathic remedies it will be necessary to mention *aconite* for the acute stages, as many of the early symptoms correspond to the indications for this remedy. *Bryonia,* however, is more clearly indicated as the character of the onset, the sharp and lancinating pains and the peculiar local features, point decidedly to this remedy. Not alone does this refer to the synovial effusion, but it is indicated as well for the extension of the joint inflammation. *Rhus tox.* is also a valuable remedy, as it relates more particularly to the general infection and the low grade joint inflammation; the pains seem to penetrate the deeper joint structures and are felt in different parts of the body at the same time; the limbs feel stiff and paralyzed, the pain is increased by beginning movement, but relieved by continued motion, while the slightest exposure is liable to cause an aggravation; the parts are red and swollen, tenderness is extreme and the inflammation may assume an erysipelatous character; the adynamic condition will be represented by the typhoid features and the extreme restlessness. In many respects this remedy is complementary to bryonia, while it may always be compared to baptisia and echinacea. *Arnica* is often indicated when the greater muscular involvement is represented by the bruised sensation, and the arthritic pains are worse in the evening. *Phytolacca* applies to similar conditions with shooting and lancinating pains in the shoulder muscles, together with much rigidity of the muscles every morning. *Cimicifuga* may be compared when nervous symptoms are extreme and this, with *valerianate of ammonia, picric acid* and *agaricin,* will frequently be indicated when chorea is a sequential condition. *Caulophyllum* pertains to the pains in the small joints, *belladonna* is applicable when the joints have a red, puffy swelling with the throbbing sensations which always are suggestive of this remedy. *Cinchona* will be suggested by the conditions of general debility, hemorrhage and the exhaustions which attend the chronic cases. *Hamamelis* may be compared to this remedy as well as to arnica. *Sulphur* will always have a degree of usefulness in this disease when chronic conditions antedate the arthritic attack; it is also very useful in the rheumatic fevers of children.

Ledum, though referring more particularly to the gouty patients, has a certain influence in the inflammatory cases. It is indicated in those who suffer from a sub-acute form of this disease. It is applicable when the small joints are affected as well as the larger ones, and the toxin is evidenced by a low grade skin inflammation. *Hypericum* refers more particularly to a nerve pain when injury is a factor. *Ruta graveolens,* in its action upon the periosteum and cartilages, will meet the deep-seated pains and the conditions following periarticular swellings, while *ranunculus* will correct the neuralgic seizures which affect the shorter muscles like the intercostals. *Colchicum* has for a long time been regarded as a valuable rheumatic remedy though it has recently been supplanted by its active principle, colchicin. This has been highly favored by Goodno, who gives it in an alcoholic solution, one grain to the ounce. Five drops of this may be given as often as every two hours. *Nitric acid* and *mercurius* may be indicated in cases with a specific history, *ferrum phos.* has a certain efficiency when anemia is a factor, while *kalmia* and *spigelia* will always be of use when the chest muscles are involved, or pseudo-anginal attacks occur. *Eupatorium perf., gelsemium, pulsatilla, rhododendron, natrum sulph., natrum mur.* and many other remedies will be suggested by their cardinal symptoms in certain cases.

The *diet* in the treatment of rheumatic fever is always of importance. During the febrile stage it should, of course, be of liquid character, limited and given at regular intervals. Meat broths should not be employed to a great extent, and when a solid diet is resumed,—after the febrile excess,—the proteid articles should be sparingly resumed. *Diuretic action* is always necessary when there is any renal insufficiency, but this object is usually attained without the aid of medication, when a sufficient amount of water is administered. Basham's mixture, diuretin, urotropin and potassium citrate are standard agents for this purpose. The *bowels* will also require careful attention and in case of constipation saline cathartics will be of best service. Calomel is rarely demanded, preparations of cascara usually being sufficient. Elaterin (gr. 1/20 to 1/10) may be given every two or three hours, until a copious watery movement is obtained. Potassium bitartrate, in 30 to 60 grain doses, is highly recommended by Dr. F. W. Wood. *Euonymin, iris versicolor, chinoidin, chionanthus, chelidonium* and *carduus* may be of service in this respect.

GONORRHEAL ARTHRITIS.

This is a septic synovitis following joint invasion by the gonococcus. It appears usually during an acute gonorrheal attack and may be incident to any stage of that infection.

Pathology and Symptoms.—These are characterized by an acute synovitis affecting the knee, ankle, shoulder, wrist and odd joints like the sternoclavicular and temporomaxillary. A periarticular invasion affecting the ligaments and tendon sheaths, especially the wrist and hand, may be observed. The gonococcus may be found in the effusion or in the periarthritic exudate, while in the purulent forms there may be a mixed infection with staphylococci or streptococci. The disease occurs far more frequently in men than in women. *Acute gonorrheal arthritis* is characterized by the sudden involvement of one or more joints and the evidence of an apparent fibrinous or serofibrinous effusion. Pain is usually severe and the swelling may be extreme if the periarticular structures are invaded. Temperature may be observed, though it may pursue an obstinate course without much constitutional disturbance. The exudate may undergo resolution if the inflamed joint is given absolute rest. Should suppuration supervene, the result is less hopeful. The disease may assume the polyarthritic type, the knee, wrist and ankle generally being affected at the same time. Usually, however, there is a later concentration of the inflammatory invasion, and one joint, generally the knee, is particularly affected. In rare instances the bursae, tendon sheaths, and the periosteum are affected. Sometimes the gonococci invade the blood, giving rise to an intense septico-pyemia. Suppurative arthritis may then take place and acute endocarditis may be a marked feature of the symptomatology. Pericarditis, pleurisy and iritis are also liable to exist under this general infection. The *chronic form* of gonorrheal arthritis may follow, the acute manifestations appearing usually as monarticular, the knee being most frequently affected. In these the acute symptoms have disappeared, though the extensive swelling may cause much pain and discomfort. All forms of gonorrheal arthritis pursue an obstinate course and a certain degree of ankylosis may persist.

Diagnosis.—This depends upon the history of a gonorrheal infection, and the presence of gonococci in the joint effusion, or more rarely in the blood, together with the absence of the characteristic signs of other joint diseases.

Treatment.—Internal remedies give little relief to the joint involvement, though they may be employed for the relief of the febrile symptoms which may sometimes arise and the general destruction of the gonococcus. Aside from such homeopathic remedies as *arsenicum iodid, iodoform, hepar sulphur, sulphur, silicea* and *calcarea fluori~~um~~* relief is obtained by the use of 1 to 2 grain doses of methylene blue, four times daily. The arthritic treatment, however, is purely surgical. The inflamed joint must be given proper rest, a splint often being employed for this purpose. Wet boracic dressings will give some relief. It is best to pierce the synovial membrane with a small trochar and irrigate the inflamed joint with a 2 per cent carbolic solution under strict aseptic precautions. When the inflammation subsides massage and passive movements must be undertaken.

DIPHTHERIA.

Diphtheria has been described under several different terms, membranous croup being the most frequent synonym. It is an acute contagious disease caused by a specific bacterium,—the Klebs-Loeffler bacillus. It is characterized by the formation of a false membrane on the oral, faucial, nasal, laryngeal, tracheal, bronchial or other mucous membranes and by secondary constitutional symptoms. Pseudo-diphtheria is a term used to describe that class of cases which is characterized by the formation of a false membrane not caused by the Klebs-Loeffler bacillus. It is probably of streptococcic origin.

Etiology.—There is at present no doubt that all cases of true diphtheria are caused by the Klebs-Loeffler bacillus. This organism is non-motile in character, slightly bent, with rounded ends, 2.5 to 3 microns in length and 0.5 to 0.8 microns in thickness. The cultures in blood serum appear as small, round, grayish-white colonies. The bacillus may be developed in all the culture media, but it is destroyed by a temperature below 68° F. or above 122° F. It is the production by this microorganism of a toxalbumin which creates the constitutional symptoms of the disease. Other pathogenic bacteria are frequently associated with the bacillus of diphtheria, and chief of these is the streptococcus pyogenes, while the staphylococcus albus and aureus, the micrococcus lanceolatus and the bacillus coli communis are occasionally a part of the infectious cause. The diphtheroid infections may be due to any of these, though a particular organism, the bacillus xerosis, has been described as the cause of the pseudo-diphtheritic attacks. This is identical in appearance, growth and culture development with the true bacillus, but it causes no local lesion or extreme constitutional symptoms, and, therefore, has been regarded by some as an attenuated form of the diphtheria bacillus.

The *method of infection* may be through the direct conveyance of the bacillus in a piece of diphtheritic membrane which has been expectorated, by inhaling air which contains bacilli and probably by fomites. Toys, books, clothing, drinking cups and many other articles which are handled may, therefore, be media for the direct conveyance of the bacillus. Domestic animals no doubt are frequently the means by which the infection is conveyed to children in many instances. The bacillus may live for a long time in the healthy throats of immunes and then be conveyed to one exposed by the friendly kiss. The local conditions favoring an infection must always exist, and thus a lesion of the mucosa or a catarrhal inflammation naturally favor the germ growth. A general physical depreciation is also a factor which favors the infection. Some claim that the bacillus enters the blood through the respiratory system, the constitutional symptoms thus being manifested first and the local exudate being a secondary development. The *site of infection* is usually the mucous membrane of the fauces, though other membranes as well as skin abrasions may be infected. The membrane develops at the point of infection, but the systemic intoxication is due to the absorption of the toxins into the lymphatics and blood-vessels directly from the local lesion. The life of the bacillus outside of the body may be prolonged for some time.

There are also *predisposing causes* which favor the reception of the bacillus, and the first of these is *age,* inasmuch as we find most of the cases in young children. It must not be presumed, however, that adults, or even old people, are exempt. *Sex* is a factor of no importance; *season* may possibly predispose, as we observe most cases in winter or spring, and *climate* has some influence, as the disease prevails most where humidity is greatest, and, therefore, the temperate zones furnish the majority of the cases. *Unhygienic* surroundings must also favor the contagion, for where the general health of the neighborhood is at a low standard we may find the most serious epidemics. Just what relation defective sanitation, decomposing organic matter, etc., have to the direct development of this disease can not be clearly stated. It is evident, however, that many cases seem definitely related to such conditions, and, therefore, it is proper, for the sake of a community's health, to regard these conditions with apprehension. *Immunity* is not gained by one attack, but the danger is certainly limited.

Pathology.—The characteristic pathologic feature in true diphtheritic inflammation is the *fibrinous exudate,* which is most frequently found on the faucial mucosa, being superficial in mild cases, but in more severe cases involving the mucosa and even the submucosa in the coagulation necrosis. *Hyperemia and edema* of contiguous tissues, together with the appearance of muco-purulent secretions, are also evidenced. The color of the exudate is at first yellowish-white, but it soon assumes that dirty, grayish-yellow color which, once seen, can not be mistaken a second time. It usually gives to the breath a characteristic foul odor. The membrane is properly considered as a coagulation necrosis, and its production is described as follows by Tyson: "The diphtheritic poison, probably admixed with fibrin from the blood, infiltrates the wandered-out leukocytes and the epithelial cells of the part, especially the more superficial, causing first their death and then a hyalin transformation and simultaneously coagulation. The result is a plate of necrotic tissue and coagulated fibrin and, hence, the word 'coagulation-necrosis.' The *membrane* is thick and firmly attached so that it is difficult to remove it, and bleeding generally attends this act. The edges are usually thin and irregular, though they may be elevated and wrinkled when the membranous growth is arrested. In the midst of this organic membrane and necrotic tissue the *diphtheria bacilli* are found. The extension of the diphtheritic process may go as far as the bronchi and, in rare instances, the esophagus and stomach have been invaded. A lobular pneumonia or a generalized bronchitis may be associated with the constitutional symptoms and a serious pneumonia from a mixed streptococcic and pneumococcic infection may also co-exist. The *pseudo-diphtheritic membrane* consists of fibrin, pus, disintegrated leukocytes, epithelium, a few bacilli and sometimes blood corpuscles.

The *heart* undergoes fatty degeneration in severe cases, while the mild cases of diphtheria will produce only a slight parenchymatous cardiac change. Endocarditis, pericarditis and interstitial myocarditis are occasional sequences of the disease. The *pulmonary complication,* which causes the most deaths, is broncho-pneumonia, arising from secondary infection. The *spleen* and *liver* may be slightly enlarged and they will also present the degenerative lesions of acute infections. The *kidneys* will show degeneration, as the result of toxin absorption, which will vary from a slight degeneration to a severe nephritis. In older patients the nephritis may be of the interstitial or glomerular character. The *nerve structures* also undergo pathologic changes, the most important of which are the parenchymatous and interstitial lesions. These perversions will exist in those nerves which are concerned in the paralyses which follow the disease. The nerve fibres, supplying the throat muscles, will be most frequently affected, and a round cell infiltration or a fatty change will be usually discovered here. The cerebral nerve fibres may also be subject to similar changes. The *cervical lymphatics* are frequently enlarged, but rarely suppurate. The *blood* shows some changes, its coagulability being diminished, the red corpuscles are slightly decreased, and the white corpuscles increased—in severe cases to the point of extreme leukocytosis. The *general infection* is manifested by the usual signs of septic intoxication and this is one of the significant features of the disease.

Symptoms.—The *period of incubation* varies from two to ten days, being shorter in the virulent cases. The prodromal signs are not always suggestive of the serious infection and the slight complaints of malaise may be very misleading. The *invasion,* in the simple tonsillar diphtheria, starts with a slight febrile attack, which may be preceded by a sense of chilliness and aching in the limbs and back. In severe cases in young children a *convulsion* often initiates the attack. The fever gradually, or in severe cases rapidly, increases in gravity, the child becomes listless, and even stupid, and loses its appetite. The urine contains a small amount of albumin while examination will reveal a faucial and tonsillar exudate and the angry swelling of adjacent tissues.

Pharyngeal diphtheria, aside from the similar symptoms of the incubation and invasion periods, will be detected first by the child's complaint of sore throat and the peculiar lividity of the mucous membrane, which is noticed at the early examination. The *membrane* usually appears at first on the tonsils, simulating, to a great extent, the exudate of follicular tonsillitis. The membrane spreads very soon from the tonsils to the pillars of the fauces, the soft palate and the posterior wall of the pharynx, the swelling of these tissues preceding the appearance of the exudate. The membrane changes soon from a yellowish-white color to the dirty gray color which is so characteristic. It is firmly adherent and its removal causes bleeding. The *glands* beneath the angle of the lower jaw and in the neck are soon enlarged and tender. The *constitutional symptoms* are now marked and the child is extremely sick, and while the temperature shows only a moderate rise, the pulse is rapid and thready, indicating a profound toxemia. If the urine is now examined, albumin will invariably be found. In mild cases the pseudo-membrane may clear up in a few days, the temperature and systemic intoxication will disappear and the child rapidly recovers. In marked cases of true diphtheria, unless antitoxin and other appropriate remedies are used, there will be an aggravation of the constitutional symptoms, an increase in the temperature will be noticed, delirium will generally appear and the child may go into a deep coma in which death soon ends the suffering. Rotch lays stress upon three conditions: the virulence of the bacteria, the local resistance and the general resistance.

In *malignant diphtheria* the rapid onset, the early development of the membrane, the pronounced cardiac failure and profound asthenia, the vomiting, the high temperature, the extensive swelling of the tonsils, soft palate and uvula, and the gangrenous appearance of the exudate, will not be misunderstood. When the temperature reaches 103°-105° F., the cardiac exhaustion will be pronounced. The pulse then becomes very feeble, the urine may be suppressed or highly albuminous, the face becomes edematous and cyanotic, an erythematous or purpuric rash may appear on the face and neck, coma may follow, death taking place in a few days after the appearance of the initial symptoms.

Nasal diphtheria may exist in conjunction with or independent of pharyngeal diphtheria. In some instances the presence of a nasal membrane may not be observed, a purulent or bloody discharge with obstructed respiration being the only manifestations. These cases may show a chronicity and the contagion may be conveyed to others by close association. Nasal diphtheria may, however, assume a malignant form, in which the membrane is liable to extend into the nasal orifices, giving rise to serious local complications, such as purulent conjunctivitis and otitis media. The constitutional symptoms, in such instances, are also severe, death usually taking place with coma.

Laryngeal diphtheria or membranous croup is a serious and deceptive form of the disease. The membrane may be seen with a mirror as a slight exudation upon the upper part of the larynx, or it may be an extension from an apparently innocent pharyngeal exudate. The early signs of constitutional invasion are not marked, and, hence, the physician is liable to be deceived as to the gravity of the disease. A marked hoarseness and a rough or metallic cough will indicate the croupy condition, but all of a sudden these symptoms will become aggravated to the extent that the breathing is paroxysmal and difficult until the accessory muscles of respiration are called into action and this terrible condition of dyspnea is soon followed by cyanosis. Death will take place as the result of suffocation unless prompt relief is given by antitoxin, intubation or tracheotomy. In some instances a severe cough will disengage a portion of the membrane and this will give at least temporary relief. The temperature may not be very high and the toxic symptoms may be absent on account of the location of the lesion.

Wound diphtheria takes place when the bacilli enter a bruise or a break in the cutaneous structure. Operations may also be contributing local factors and granulations always favor an infection. The *systemic infection* in all forms of diphtheria is a serious phase of this disease which many times is not recognized when the local lesion is apparently slight.

The *complications* have already been referred to, bronchopneumonia, albuminuria, dysphagia, cardiac exhaustion and otitis media being the most serious. There are, however, many sequelae which may possibly be overlooked. *Anemia* may persist for a long time and require constant medical attention; *chronic naso-pharyngeal catarrh* is another disturbing symptom and *postdiphtheritic paralysis* may affect the muscles of deglutition, the ocular and ciliary muscles, or it may involve the limbs to the extent that locomotion is interrupted. These conditions, however, are usually amenable to remedies and the use of electricity. A single or *multiple neuritis* may continue for some time, and this will be an obstinate sequela. *Cardiac paralysis,* due to involvement of the cardiac nerves, may give rise to a troublesome tachycardia or bradycardia, terminating often in sudden death by overexertion.

Diagnosis.—The diagnosis may be positively made by bacteriological examinations whenever it is possible to get a piece of the exudate or swab the suspected parts. Our present day opportunities for making or getting the culture reports are within the reach of nearly every practician. A careful physician who has had any experience with epidemics of diphtheria will be able to make a comparatively safe diagnosis from the symptoms already outlined. It is best, however, in every doubtful case, for the sake of the patient, to err on the safe side by diagnosing every suspicious case as true diphtheria. Treatment, now accepted as correct for this disease, may well be given for spurious cases rather than to take any chances. Our greatest danger will exist in the masked cases which we may possibly overlook. With bacteriological opportunities at hand there should be no excuse for a mistake. When a child presents sore throat with spreading patches, glandular enlargement, moderate rather than high temperature, fetid breath and drowsiness, together with a weak and rapid pulse, it is safe to make a diagnosis of diphtheria and treat it as such. A *differential diagnosis* may be made in many instances by a careful study of the symptoms, but we should be doubly sure by making or getting a bacteriological report. A *streptococcus infection* of the fauces or pharynx is frequently met with and simulates closely true diphtheria. Differentiation can be made, however, by the following facts: In the former the temperature is higher, the breath is not usually fetid, nor the toxemia so marked. The exudate, while it tends to spread, does so slowly and very often remains stationary. If in these cases antitoxin is used there is not a disappearance of the exudate and constitutional symptoms as in true diphtheria. *Follicular tonsillitis* may be absolutely differentiated by bacteriological examination. We may, however, establish the diagnosis clinically by the following points of difference: In tonsillitis the membrane is usually confined to the tonsils, showing in the early stages the exudate coming from the tonsillar crypts, while in diphtheria the membrane covers the tonsil and *spreads* to the fauces and uvula; in diphtheria the membrane has a more dirty-yellow color and bleeding takes place when it is detached. In tonsillitis the febrile manifestation has a more sudden onset, and the temperature—for a day or two—may be much higher than in diphtheria. Albuminuria is rarely present in tonsillitis and the disease runs a short course. *Scarlet fever* will usually offer but little difficulty in diagnosis. The rash is usually present and is generally typical. A similar rash may appear in diphtheria, but generally this has an erythematous character. Later the desquamation will positively settle the diagnosis.

Prognosis.—Epidemics of diphtheria, before antitoxin was discovered, were feared more than those of any other infectious disease. It is safe, however, to say that, with the early and heroic use of this agent, we may treat this terrible disease with a very low mortality. The complications and sequelae—depending, of course, upon the virulence of the epidemic and the vital resistance of the patient—will demand serious consideration, but even these are minimized by the use of antitoxin. Much, however, depends upon the attention and the capability of the attending physician, for there are many features of this disease which need care and treatment, aside from the specific infection. Cases of *pseudo-diphtheria* will always yield to symptomatic treatment, but a bacteriological examination will greatly relieve the physician's anxiety.

Treatment.—The prophylactic treatment of diphtheria is of great importance. Isolation in all suspected cases must be rigidly enforced. Cultures should be frequently taken from the throat, particularly of school children when an epidemic is raging, and this will apply also to those who have been exposed or who have the care of a diphtheria patient. One examination is not sufficient, for we must remember that the diphtheria bacilli may remain in healthy throats for a long time, without active development. It is never safe, during an epidemic, to wait for positive symptoms in those who have been exposed. The mouth and nose should be frequently cleansed and antiseptic solutions should be freely employed. Attention should also be directed constantly towards maintaining the general physical resistance lest one become predisposed to the infection. The convalescence will require constant care, not alone for the suffering patient, but from the fact that bacilli may remain in the throat of a sufferer long after the cure is apparent and thus engender a constant danger of exposing others. Emphasis should be given to this suggestion inasmuch as everyone is liable to be careless in handling the milder cases.

The *antitoxin treatment* is, to my mind, our most reliable form of treatment, and, therefore, it is my belief that it should be promptly and thoroughly employed in every suspected case from the very beginning. It is true that many in the profession have been slow to accept this specific agent, but the general consensus of opinion among experienced and unbiased practicians is at present positive as to its early use. There are many physicians who, no doubt, feel that they can manage this terrible disease with their internal remedies and local antiseptic treatment. Due credit should be given to those who are capable of doing this, but to me it seems a dangerous risk not to use antitoxin, and I always fear that the favorable reports of such treatment which are recited in our medical societies, are not always substantiated by a careful bacteriological proof that the cases were really diphtheria and not diphtheroid. We need to be very frank in this respect, for our honest aim should be to save our patients without any bias in favor of past treatment. It has been my experience that those who report unfavorable results from the use of antitoxin either did not use it early enough or in sufficient dosage, or neglected to repeat it when it was needed. Hospital experience in the management of the most severe cases certainly proves beyond doubt that antitoxin is a specific on which we may rely. It is clearly settled in my mind that many of those malignant cases which heretofore have resisted all other forms of treatment respond to the prompt and thorough use of antitoxin. With this opinion established, at least to my satisfaction, I do not recognize the necessity of an extended discussion relative to any other method of treatment. If a therapeutic principle is truly demonstrated, it should become a part of the armamentarium of every physician without prejudice. It certainly is not antagonistic to the law of similars.

The *amount of antitoxin* which should be used must be settled by the requirements of each individual case. Much discussion has taken place in this regard. The consensus of expert experience has established the fact that it must be given in sufficient dosage to secure antitoxic effect. For this purpose not less than two thousand units of the standard strength should be administered at the first instance. Some claim that this is not enough even in children and many expert practicians give as high as four to six thousand units at the first treatment. The dose should be repeated, according to indications, in six to twelve hours. In many cases when the case is seen in the first stages the initial dose is sufficient for twelve to twenty-four hours. So much has been written for and against the use of this specific agent that it seems to me unnecessary to say more on this subject except to repeat the caution that antitoxin should be administered early and in sufficient dosage. For prophylaxis in the case of those exposed anywhere from two hundred to five hundred units may be used, according to the age of the patient.

Local applications are of use in preventing general infection. Peroxide of hydrogen (50 per cent solution) may be used as a gargle or in the form of a spray. Dobell's solution, permanganate of potash, carbolic acid preparations and similar adjuvants have been used with success. A hot soda solution will often be all that is essential for this purpose, and it usually gives prompt temporary relief.

The *homeopathic remedies* have a sphere of action in this disease which can not be overlooked. While I accept the specific treatment with antitoxin I am equally partial to the indicated remedy for intercurrent conditions and sequelae. No specific agent alone will answer the purpose in every case. Remedies must be applied for a condition represented by a symptomatology, and this may refer to features apart from the specific poison. Many of our early-day practicians successfully cared for this disease before antitoxin was discovered, and it is possible to do so now, but it seems to me that the risk is too great and we should not hesitate to accept a safe specific expedient. *Mercurius, phytolacca, arsenicum iodid, arsenicum, kali bichromicum, kali mur., rhus tox, chlorid of lime, arum triphyllum, ailanthus, echinacea* and many similar remedies will have a wide sphere of usefulness in the treatment of this disease.

VARIOLA.

Variola is an acute contagious disease characterized by a fever and eruption with its four definite stages of papule, vesicle, pustule and crust. In some cases cutaneous and vesicle hemorrhages are rare symptoms.

Etiology.—The history of this disease is supposed to antedate the Christian era, and its ravages in this and European countries are sufficiently known to warrant the omission of any historic facts regarding it. Our bacteriological knowledge pertaining to this disease is not complete, inasmuch as no one up to the present time has succeeded in isolating the specific micro-organism. Roger and Weil believe they found specific bodies in the pus and blood, Funk discovered a protozoon, the sporidium vaccinale, while Councilman has recently claimed that the disease could be traced to the protozoon cytoryctes variolae. That it is truly an infectious disease is well established, for no better evidence of contagion ever existed than is manifest by an outbreak of small-pox. The infection is probably conveyed by particles of the dried scabs being carried through the air to the healthy. The exhaled air, as well as the secretions and excretions of the body, are also factors in its dissemination, since the specific poison exists in the blood as well as in the pustules and scabs. The vitality of the contagion is evidenced by the fact that it may remain dormant for a long time in clothing and furniture, and then cause an infection. All ages, even the fetus in utero, are susceptible, and no race is exempt, while the colored race and people of inferior type in civilization are more liable to suffer from its ravages. The conditions favoring the reception of the infection are, without doubt, found in a state of lowered physical vitality, as well as faulty nourishment and unhygienic surroundings. It is probable, however, that any one who has been exposed is liable to contract the disease. One attack generally furnishes immunity, while vaccination is our best means of prevention. Various opinions have prevailed relative to the period in which the disease is most contagious. While it is probably infectious during any stage, the danger is more pronounced during the suppurative and desquamative stages, and less so during the initial period.

Pathology.—The morbid anatomy of this disease embraces the eruption which is defined under the symptomatology, the hemorrhagic infiltration and the complications which may occur. The pustule starts in the rete mucosum, a coagulation necrosis taking place in its center, while the surrounding reticular spaces are crowded with serum, leukocytes and fibrin. If this inflammatory process does not extend very deep, healing may take place without any scarring. When the papillae of the true skin are destroyed a pit remains after the disappearance of the eruption. The spleen may become hardened, while a cloudy swelling may affect the cells of the liver and the kidney. Nephritis may appear as a secondary affection, myocardial alterations are not uncommon and endocarditis or pericarditis are occasional complications. Catarrhal inflammation of the larynx, bronchi and lungs may appear during the course of the disease, and hypostatic congestion is often a serious complication.

Symptoms.—A period of *incubation,* lasting, according to the receptivity of the individual, from seven to twelve days and characterized usually by insignificant symptoms, may precede the *onset,* which generally appears suddenly with a pronounced chill or a chilly sensation. This is attended by violent muscular pains in the back or by real convulsions when children are affected. *Headache* is intense and the *fever* develops rapidly, the temperature rising as high as 104° or 105° F. in the first few days. The *pulse* is rapid, though hard and strong, the *respirations* are also accelerated, bronchitis is often present, the tongue is coated and gastric disturbances are prominent. The patient is restless, delirium is often observed and a state of stupor may exist at this period. The *initial rash* appears about the second day, and this may assume a form which resembles a diffuse scarlatinous rash or a macular form, which resembles the eruption of measles. This eruption may have a hemorrhagic character, purpuric spots appearing on the abdomen and thighs. The stage of invasion lasts about three days.

The *characteristic eruption* appears on the third or fourth day, first upon the forehead and then extending rapidly over the face, scalp, hands, wrists and entire body. In character it is *macular,* like measles, light red in color at first, but later it becomes dusky red. In twenty-four, or at least forty-eight, hours the maculae develop into distinct *papules.* These feel like shot underneath the skin and are attended by much itching and burning. On the fifth or sixth day the apices of the papule contain liquid, and, as the entire papule becomes filled, *vesicles* with marked central umbilication are formed. By the eighth day the turbidity of the vesicular contents becomes bright yellow, the umbilication disappears and the *pustule* is established. Each pustule is usually surrounded by a red border unless the eruption is confluent when the pustules coalesce. The parts as a whole are greatly swollen, the face particularly showing the disfiguration. Pain is often a marked symptom on account of the tension. When the pustules are not confluent the desiccation begins on the tenth or eleventh day, and by the fourteenth day the papules are converted into crusts, which soon drop off, becoming only a discoloration, in mild cases, or in severe cases pits or scars, which finally fade into white spots. Invasion of the *mucous membranes* by the eruption is a simultaneous feature of the symptomatology, the mouth, tongue, soft palate, pharynx and nares being the most frequent parts affected. Pustules have also been found in the esophagus, trachea, bronchi, and even in the mucosa of the gastro-intestinal canal. When the mucous membranes are affected ulcers, as the result of maceration of tissue, supplant the pustules.

The *fever* of variola is a characteristic one. The temperature, succeeding the chill, rises rapidly to 103°-104° F. with some evening exacerbation. When the papules appear there is either a remission or a complete disappearance of the temperature. When the pustular stage is reached a secondary fever, which is suppurative in character, is usually experienced. This will be mild or severe, in accordance with the severity of the disease and the extent of the suppuration. It generally lasts three or four days, until desiccation takes place, and then it disappears by lysis. Any septic complication will give rise to a continuation or a recurrence of the secondary fever. Other special symptoms will exist in accordance with the character of the complications.

Modified forms of the disease often exist. The *confluent* variety was more common in early epidemics than at present. In this the symptoms of the invasion are more severe, the eruption appears earlier, the papules are more numerous and when the pustular stage is reached the parts involved by the eruption present a continuous suppurative area. The facial disfigurement is, therefore, extreme, and this is often enhanced by the septic involvement of the cervical lymphatics. The temperature attains a higher degree than in the discrete type, the nervous symptoms are accentuated, and, as the typhoid state is liable to ensue, the patient may die in a condition of septic coma. *Hemorrhagic small-pox* is recognized in two forms: The first is called *purpura variolosa,* or black small-pox, which is usually fatal, the hemorrhagic symptoms appearing very early. The constitutional symptoms are pronounced and in the evening of the second day the hemorrhagic rash is recognized by small punctiform hemorrhages, which appear in the groins, the face and other parts of the body. The rash becomes more extensive and decidedly more hemorrhagic in character in a very short time. Conjunctival ecchymoses and mucous membrane hemorrhages appear as early as the third day, and death may take place at this time. Should the patient survive longer the skin assumes a purple or plum color, and this, with the increased conjunctival and mucous membrane hemorrhages, creates a frightful facial disfigurement. Hematuria, hematemesis, melena, hemoptysis and even metorrhagia in females may add additional severity to the symptoms. The pulse is rapid, small and hard, the respiration is quickened and death may occur as early or even earlier than the sixth day. The other form of hemorrhagic small-pox is known as *variola hemorrhagica pustulosa,* the hemorrhage being noticed in a severe case of variola vera during the vesicular or pustular stage. While the hemorrhagic phase of the eruption is always a dangerous condition, this variety is not so alarming as the black small-pox unless it appears very early. If the bleeding takes place about the time of maturation, recovery is possible. Mucous membrane hemorrhage is a common feature in this form, and, of course, this adds to the gravity, death usually resulting as early as the seventh or eighth day. Variations in the virulence may take place in all of these forms in different epidemics, one form of bleeding, during the vesicular stage being particularly mild. An *abortive form* has been described, in which a speedy recovery occurs after the subsidance of intense invasion symptoms.

Varioloid is a modified or light form of the disease which affects those who have responded to vaccination. The initial symptoms are mild, the papules appear as early as the third or fourth day, and are few in number, being confined to the face and hands. The fever is slight and short in duration, no secondary temperature being recognized. The vesicular stage is not marked and pustules rarely appear. The affected parts do not usually show any signs of scar tissue, though, in exceptionally severe cases, all the extreme symptoms of the disease may be present. In these cases the rash sometimes assumes a "horn-pox" appearance, the papules becoming dry and hard about the fourth or fifth day. In some instances varioloid may exist in young patients without any sign of eruption.

The *complications* which are frequently associated with variola are liable to be as serious as the disease itself. Laryngitis, with a fatal edema of the glottis, may induce sudden death, while broncho-pneumonia, parotitis, diarrhea, vomiting and albuminuria are conditions always to be watched for. Cardiac complications are fortunately rare, though myocarditis with endarteritis of the coronary vessels is sometimes a serious sequence. Nervous complications are often the cause of much concern. In children convulsions may occur, while in adults the delirium is liable to become violent and persistent, or it may precede a fatal coma. Insanity is a rare sequela and a multiple neuritis may give rise to a pseudo-tabes. Arthritis may appear during the period of desquamation, and in rare instances this may be followed by acute necrosis. In the convalescent stage boils and painful acne are often troublesome, and conjunctivitis, though uncommon in modern practice, is sometimes an obstinate complication.

Diagnosis.—Recognition of the disease is helped usually by the knowledge of a prevailing epidemic and the characteristic eruptive stages. To an inexperienced practician, however, many difficulties will arise in the differentiation of similar eruptive diseases in their early stages. *Scarlatina* will only be confusing in the early stages when it resembles the initial rash of variola. Scarlatina, however, has a more intense and a universally distributed rash over the entire body, and does not present the papular, vesicular or pustular stages. *Measles,* in its macular stage, will resemble small-pox, and many errors have been made in this differentiation. Measles, however, has none of the severe initial muscular pains, while the coryza and the peculiar cough are decidedly suggestive. In measles the fever may rise with the eruption, while in small-pox the primary temperature subsides as the eruption appears. The absence of the shot-like feeling which is so distinctive of variola is also a marked differentiation in the early stages, and to an experienced observer there will be no further doubt after the measles eruption is well established. Should hemorrhage be a factor in this disease or in scarlatina the doubt, of course, will, for a time, be enhanced. *Cerebro-spinal fever,* when purpuric spots are present, will offer many simulating symptoms, but the retraction of the head and the attending rigidity are generally sufficient to establish the diagnosis and further doubt will usually be removed by a microscopic examination of the cerebro-spinal fluid. *Impetigo contagiosa* has no initial symptoms resembling small-pox, the eruption is vesiculo-pustular, superficial, and the individual lesions are more extensive than those of variola. *Pustular syphilides* are not attended by the same febrile symptoms, the pustule has an indurated base, the eruption assumes various forms, the color is coppery and neither umbilication nor pitting takes place. The history of a primary lesion is also a confirming factor. *Varicella* will cause the greatest errors of diagnosis in mild epidemics of small-pox, though it is usually confined to children and may appear in patients who have been vaccinated. The period of invasion is usually shorter, the onset is less intense, and a prodromal rash is rarely present. The rash may be more abundant on the body, the papules are not "shotty," the vesicles are more superficial and rarely umbilicated, and the "pocks may be seen in all stages of development side by side." In small-pox the vesicles are multilocular, while they are invariably unilocular in varicella. In the latter disease a secondary fever rarely occurs. In small-pox the *blood examination* will reveal a leukocytosis, "the large mononuclears being increased from 4 to 10 per cent and the myelocytes from 2 to 10 per cent. In the hemorrhagic form nucleated red cells may be found."

Prognosis.—The milder epidemics which seem to prevail at the present time, together with the better general care of the patient, has reduced the mortality decidedly. The severe cases of the confluent variety and the hemorrhagic forms are always to be feared. The young are more liable to succumb, while dark-complexioned peoples usually have a more severe form of the disease. The gravity of the disease is enhanced by intemperate habits and general physical debility. Complications are always unfavorable conditions, and the extreme initial symptoms are suggestive of a fatal outcome. High temperature, delirium and coma are unfavorable phases.

Treatment.—The treatment of this serious disease has undergone many modifications during the past few years, and the old and barbarous methods which once prevailed have, fortunately for the sufferer, been abandoned. *Prophylaxis* is a requirement which no physician should neglect. The proper authorities should at once be notified and the patient should be safely isolated. Those who have been exposed should be immediately vaccinated, as this procedure not only modifies the attack should one be infected, but it is a means of general protection as well. Today few of those who are exposed are liable to contract the disease when prompt and successful vaccination is accomplished. The *general management* of the case will contemplate all the requirements of modern febrile treatment, and the diet should be of liquid character in the early stages, as well as light and limited during the period of convalescence. The room should be well aired and "comfortably cool." Fresh air is an essential which should not be overlooked. The cool sponge bath should be employed the same as in the treatment of typhoid, and the bowels should be regulated by cathartic or by high salines, as may seem best in individual cases.

Local applications for the care of the eruption are not so generally employed as in the past. Thus the pastes and caustics, as well as the punctures of the pustules, have been practically discarded. It may be well to protect these from the extreme light; and cold water, containing a mild antiseptic may be applied on well-saturated pieces of lint. The wet dressing may be maintained for a longer period without renewal by applying a piece of oiled silk external to the lint. When the crusts form, moisture may be maintained by the frequent application of olive oil or glycerin, which also relieves the intense itching to a certain extent. Recently the following treatment has come into vogue. It consists in painting pure carbolic acid over a limited affected area with a camel-hair brush, applying the acid to each individual vesicle, care being taken not to touch the intervening healthy tissue. It is important to encourage the early removal of the scabs by the use of oil applications. Care of the inflamed parts must be continued assiduously during the convalescent stage to avoid scarring. The eyes will need frequent irrigation with collyria to avoid serious sequences. The complications will be met in the same manner as in other diseases; laryngeal edema often demanding tracheotomy, cardiac weakness calling for stimulants, and broncho-pneumonia requiring specially indicated remedies. The old custom of giving a teaspoonful of vinegar three or four times daily is still a favorite procedure on the part of many modern practicians.

Homeopathic physicians have been very successful in their treatment of this disease, and this is due to the fact that they have depended largely upon their indicated remedies. *Aconite* has been a serviceable remedy in the initial stage, as it corrects the restless condition and the peculiar characteristics of the fever at that period. *Belladonna* and *rhus tox* have likewise been useful, inasmuch as they successfully meet the symptoms of congestive character or the toxic manifestations of a low-grade fever. *Veratrum viride* has a decided influence in the cases of high temperature with increased vascular tension. *Gelsemium* covers many of the symptoms of the early stage, particularly when the terrible backache and muscular pains are extreme.

When the *eruption appears* we shall have an opportunity to use many of our homeopathic remedies. *Apis mellifica* will apply to the initial stage, when the burning urticarial rash has a peculiar stinging sensation, and it will be useful in the later stages, when edema about the eyes ensues and becomes general.

Croton tiglium will often be of use when the vesicles develop, and it may frequently be continued through the pustular stage. *Vaccininum* has been used extensively by practicians of our school, and many good results are reported as a result of its employment. *Tartar emetic* has probably been advocated more frequently than any other remedy included in our pharmacopeia. The general consensus of opinion seems to indicate that it should be given in the early stages rather than at the period of vesicular or pustular development, though it is naturally indicated with these symptoms. *Thuja* was recommended by Benninghausen as an important early remedy, but little has been written upon this remedy of late. *Sulphur, hepar sulphur, silicea* and *mercurius* have been suggested for the early eruptive stage. In hemorrhagic cases we may expect results from the use of *secale, hamamelis, nitric acid, phosphorus, crotalus* and *lachesis.*

VACCINATION DISEASE.

Vaccinia is an infectious disease which follows from the inoculation of man with the virus from cow-pox. This is characterized by the local pox and the mild constitutional disturbance which usually affords protection against variola. Without undertaking the various theories as to the nature of vaccinia, the methods of lymph preparation or the technique of vaccination, it is perhaps sufficient to state that vaccination is our only safeguard against the spread of small-pox, and it should be employed by all who practice medicine. It is perhaps best to vaccinate infants a few months after birth, while this precaution should not by any means be delayed longer than puberty. Revaccination should be instituted at least every six or seven years, or whenever there is a danger of exposure. The fact that vaccination does not "take". in several operations does not necessarily insure the patient's immunity, and for that reason physicians should be careful in their statements in that respect.

The *symptoms* of vaccination appear nearly as follows: From three to seven days after the operation a small red papule appears at the point of scarification and two or three days later a vesicle develops. By the ninth day this is fully developed and umbilicated; it contains a colorless lymph, is multilocular, surrounded by an inflamed areola and itches excessively. The surrounding inflammation and induration may vary in different cases; frequently an erythematous rash may affect the whole arm and the swelling is often pronounced, the axillary or inguinal glands partaking of the inflammation. In ten or twelve days the pustule dries up, the symptoms gradually subside and the scab develops, usually remaining on the arm for a long time. Suppuration should not take place if aseptic precautions are taken during the vaccination or the dressings afterward. Unfortunately too many patients vaccinate themselves, and it is not expected that they will take more than ordinary precautions in cleanliness.

The *treatment* of a vaccination should not be required, but when infection takes place, or when the erythematous swelling is extensive, a wet boracic dressing may be required. This is the best kind of local treatment and great precaution should be used in advising the patients against vaselin applications, etc. Internal treatment will include such remedies as *belladonna, baptisia, arnica, hamamelis* and *hepar sulphur, silicea* and *thuja.*

VARICELLA.

Varicella or chicken-pox is an acute contagious disease of children, characterized by a cutaneous eruption which is papular, vesicular and pustular, and a fever with mild constitutional symptoms.

Etiology.—It is particularly a disease of childhood, the majority of cases occurring under the sixth year. It prevails usually in the epidemic form, though sporadic cases may be found from time to time. While in many ways the disease simulates small-pox, they are not the same, inasmuch as neither furnishes immunity from the other.

Symptoms.—The *incubation* period lasts from ten to twelve days and the *invasion* may be announced by a chill or a chilly sensation, which in some children amounts to a convulsion. The eruption develops within twenty-four hours, and often this is the first expression of the child's illness. A prodromal rash rarely appears. The *eruption* is found first on the trunk, though it may begin on the face and forehead. The first sign is that of raised red papules, which, in a few hours, are transformed into hemispherical vesicles, containing a clear or turbid fluid. There is rarely umbilication or areola, and the eruption is much more superficial than that of variola. The surrounding skin does not seem to be affected by the inflammatory invasion, and there is very little edema. In about thirty-six or forty-eight hours the vesicular contents become pustular and by the third or fourth day—at the end of a week at the latest—they are converted into dark brown crusts, which soon fall off, leaving no scars, though pitting may take place if suppuration has been encouraged by scratching. Fresh crops develop during the first two or three days, so that pocks in all stages of development are present and this constitutes the marked differentiation in comparison to small-pox. The eruption never becomes confluent and the pocks are not usually so numerous as in small-pox. Sometimes the vesicles become very large and resemble bullae. Itching is usually intense, and frequently causes the child to scratch the parts until the suppurative process is encouraged to the extent that sores develop and ugly scars remain after the disease is cured.

The *temperature* is highest on the second or third day, when the eruption begins to appear. In mild cases this may be as high as 101° or 102° F., while severe cases will register a temperature as high as 104° for a day or two only. The majority of cases are mild, and these are characterized by the slight fever and the small number of eruptive spots. The temperature subsides gradually as the eruption disappears. Other symptoms are rare.

Complications may arise in certain cases, but usually these are rare. *Erysipelas* may develop in the neighborhood of the pustular pocks, and this is often a serious complication in delicate children. *Adenitis* may also co-exist in tubercular subjects, and suppuration is liable to take place in such instances. *Nephritis* is a rare but serious sequence.

Diagnosis and Prognosis.—Varicella has already been differentiated from variola, and it is not usually a difficult matter to compare this with other diseases. The appearance of the rash on the body first, its superficial character, the bleb-like appearance of the vesicles, the absence of an infiltrated area surrounding each vesicle and the different stages of the papular, vesicular and pustular development will settle the diagnosis. The prognosis is always favorable in the majority of cases, but due care should be taken during the eruptive stage against improper exposure.

Treatment.—Proper isolation, hygienic surroundings and sufficient rest in bed are the most mandatory essentials. The diet should be light, the parts should be occasionally washed with a weak carbolic solution and the indicated remedy should be given. *Tartar emetic, sulphur, hepar sulphur, croton tiglium,* as well as *aconite, belladonna, rhus tox and gelsemium,* should be considered.

SCARLET FEVER.

This is an acute contagious fever which is characterized by a diffuse scarlet eruption, with desquamation, faucitis, nephritis and other sequelae. It is usually a disease of childhood, though no age is exempt. It is endemic in cities, but it may appear in epidemics from time to time.

Etiology.—The organism which causes scarlet fever has not yet been discovered. The streptococcus pyogenes has been found in many instances, but this may be due to the complications which may attend the disease. Class claims the discovery of a diplococcus scarlatinae in the blood, throat secretions, epidermal scales and the urine of the scarlatinal patient, and he has made experiments, with partial success, to obtain an antitoxin. The virus is probably conveyed by the epidermal scales and throat and nose excretions. It has for a long time been maintained that the exfoliated epithelium was the sole agency of contagion. For this reason the disease was not supposed to be communicable except at this period and physicians have generally considered it as non-contagious before the eruption was well established. Exceptions to this, no doubt, exist, for cases are on record in which the disease has been conveyed by naso-pharyngeal secretions, even after the period of desquamation.

The *conveyance* of the organism takes place by contact with the patient, though it may be distributed by fomites. The long life of the organism makes the contagion a possibility until the desquamation period is entirely passed, and its tenacity is so great that it may remain in articles of furniture for a long time. Thus the scaly particles are freely disseminated in the air and anything which has been touched by the patient or the attendants may communicate the poison even after the patient is cured. Those who have been near a scarlatina patient should observe proper disinfection. The *infection* is usually communicated through the respiratory tract to the throat and the systemic involvement, in most cases, starts here. The alimentary canal may also be the medium for its entrance, and this has been evidenced by the epidemics occurring through milk contamination. It is claimed that the blood may convey the disease, as pregnant mothers have, in rare instances, given it to the child in utero. *Predisposing* factors are of some importance, as children under 10 years of age are most frequently afflicted. Infants are less liable to contract it, and it is of rare occurrence in adults even when exposed. Open wounds may be the media for its conveyance, and seasons have something to do with its development, for it often prevails in epidemics during the autumn and winter months. Single attacks usually bestow an immunity.

Pathology.—There is no characteristic morbid anatomy in this disease other than the typical eruption, the throat complications and the occasional sequelae. The exanthem disappears after death unless hemorrhagic extravasations are present. In rare malignant types a post-mortem eruption may be seen. Visceral engorgements may be noted in case of an early demise and ulcerative lesions may be found in the throat and adjacent structures. Visceral changes, characteristic of other infectious diseases and the pathological phases which attend the various complications are referred to under other subjects. The blood is dark and its coagulability is impaired.

Symptoms.—The *incubation* period may vary from one to eight days and prodromes are often absent. The *invasion* usually occurs suddenly, sore throat often being the first complaint. Nausea is often an early disturbance, and vomiting is a characteristic initial symptom. The pulse is strong, rapid and hard, often being as high as 120-140 in the beginning. The temperature rises rapidly to 103°-105° F., and is liable to remain high for a few days.

The *eruption* appears usually on the second day, though it may be delayed or irregular in its development. It is recognized at first on the neck and chest, but it may appear first on the abdomen or any part of the body, and from these points it covers the entire surface. The rash is characterized by its uniform scarlet color, which has given rise to the descriptive term, "boiled lobster appearance, which contrasts distinctly with the pale lips." A more careful examination will reveal the multitudinous red points, surrounded by an erythematous blush, though the erythema may be slight in some cases. The redness disappears by pressure, but promptly returns when the pressure is removed. In some instances the rash may be patchy, thus simulating measles, while a dusky color and hemorrhagic extravasations may be features of malignant cases. Vesicles with turbid contents are characteristic of scarlatina miliaris. The face, in rare instances, may show no signs of the rash. When the eruption is pronounced the burning and itching is often intense, and roughness of the skin may be determined by palpation. In a few days the rash begins to fade, leaving the skin mottled and dry. *Desquamation* commences with the abatement of the rash, usually in the order of the eruptive development reaching its maximum intensity about the tenth day. In mild cases this may be detected with difficulty, but in severe forms it may be prolonged for weeks. This is a very important period in the treatment, for the contagion is disseminated by these scales.

The *tongue* is red at the tip and edges, with the papillae standing out prominently through the coating. In a few days this coating clears, giving a clean red appearance, which, with the protruding papillae, has been termed the "strawberry tongue." The mouth, pharynx and tonsils are also bright red and sore throat is a prominent symptom. In malignant cases this may partake of the diphtheritic character. The *nervous symptoms,* which, in the beginning, may take the form of convulsions, are represented later by restlessness and delirium, which is often active when the temperature is high. The *decline,* of all symptoms in the typical case, takes place gradually as defervescence ensues. Should the fever continue longer than a week or ten days, tonsillar ulceration, infection of the cervical glands, otitis media and nephritis may be feared. *Leukocytosis* develops early and is most pronounced when suppuration ensues as the result of complications. The *urine,* in mild cases, may be scanty and high colored, with an excess of urates and uric acid.

Several types of the disease have been observed. A *mild form* occurs when the rash is the only suggestive symptom. In these cases the stage of invasion presents no symptoms and none of the subsequent features of the typical disease are in evidence. Scarlatina may also exist without eruption, and it will only be discovered by the sore throat and the strawberry tongue, together with a slight fever and the development of new cases. Nephritis may be feared in either of these forms. *Traumatic scarlatina* is that variety in which the eruption starts in a wound and spreads over the body from this point.

The *anginose form* is characterized by the severity of the throat symptoms, the swelling of the fauces and tonsils, causing marked dysphagia. A false membrane may develop on these parts and extend to the larynx, pharynx, nasal cavities, trachea and bronchi. The throat conditions and the general sepsis resemble diphtheria and frequently the Klebs-Loeffler bacillus is a factor, though the streptococcus pyogenes is the more constant micro-organism. The *malignant form* of scarlatina is characterized by the grave systemic poisoning, the initial prostration, and the intense adynamia with cardiac depression. The respiration is always accelerated, the skin is dusky, the eruption is slight and atypical, while the pulse and temperature are unusually high. Delirium is an early symptom, and death usually comes as the result of early coma and convulsions. The *hemorrhagic form* is recognized by the hemorrhagic extravasations, epistaxis and occasional hematuria. The poorly nourished children are most frequently afflicted with this form and usually die.

The *complications* which occur in connection with scarlet fever are numerous and generally severe. *Acute nephritis* is quite common and is always to be regarded with apprehension. It is a feature of the mild cases as well as the severe, and, while it is more liable to appear after desquamation is well established, it is even more severe when it develops in the earlier stages. The specific cause is, therefore, a factor, though its appearance, usually during the period of desquamation, would indicate that the increased activity of the kidneys, when the skin function is impaired, has much to do with this complication. The nephritis is generally parenchymatous in character, though it may partake of the acute interstitial variety. It is fortunate that this complication when recognized early and cared for, usually disappears. *Otitis* is a very serious and painful complication, arising from an extension of the inflammation of the eustachian tube to the middle ear. This, with all its attending features, may arise during the early stages of the disease or it may appear at any time when there is an associate pyogenic infection. *Meningitis* may arise in connection with otitis or appear as a sequence of scarlatina. Adenitis may be a complication of any form, and become very serious by extensive involvement and suppuration. *Scarlatinal synovitis* occurs in many cases during the early stages of the disease or at the time of defervescence as a result of the specific poison. Septic arthritis may also appear after a suppurative involvement of the tonsils. The true scarlatinal arthritis affects the wrists most frequently and less commonly the small joints, the elbows and the ankles. In septic involvement the knees are more frequently implicated. Typical rheumatic arthritis, with endocarditis, is a rare complication. *Cardiac complications* will depend upon the severity of the disease and the treatment which is instituted. If the patient is kept warm and in bed until the disease is absolutely cured and no stimulating drugs have been administered, we may minimize this danger. On the other hand, if a cardiac weakness pre-exists we may fear an accentuation of that trouble. Endocarditis is probably the most common sequela of this kind, though pericarditis is not rare. *Pneumonia,* either broncho or lobar, may complicate scarlatina, appearing most frequently between the tenth and fifteenth day of the disease. *Nervous affections* are commonly expected, as the disease appears most frequently at the time when the child is susceptible to chorea and other functional disorders of this kind. *Paralyses* and multiple neuritis are also possibilities, as are convulsions and hemiplegia.

Diagnosis.—In the typical case it will not be difficult to make the diagnosis, as the peculiar rash following the short period of incubation is the most distinctive feature. When the *masked forms* appear the best diagnostician will be confused. The throat symptoms, the nephritis and the detection of the scales in the bed should direct our attention to cases of this kind. The punctate character of the eruption, when slight, is often a determining factor. The differential diagnosis must take into consideration several other diseases. *Drug rashes* are often confusing, but these are rarely so diffuse as that of scarlatina. Belladonna eruption is short in duration and furnishes no constitutional symptoms. *Measles* will be diagnosed by the later appearance of the eruption, the mottled character of the rash with its irregular patches and thin crescentic outlines, the more extensive involvement of the face and the usual absence of throat symptoms. The coryza which precedes and the cough which persists are also good differential symptoms. *Rötheln,* or rubella, slightly simulates the rash of scarlatina, but it has no signs of desquamation, throat symptoms are rare, it is shorter in duration and the constitutional disturbance is slight. *Acute exfoliative dermatitis* resembles scarlatina during the eruption, but the desquamation begins earlier and involves the hair and nails. It, moreover, resembles scales and crusts, and, after it is cast off, leaves a moist surface, from which a second exfoliation may follow. Throat and constitutional symptoms are not marked.

Prognosis.—The prognosis varies according to the severity of the epidemic, the character of the disease and the vital resistance of the patient. The malignant form is, of course, serious, and the complications, particularly nephritis, are always to be regarded with apprehension. Now that its infectious nature is known, we naturally take more than ordinary care so far as diet, warmth and hygiene are concerned. Under homeopathic treatment the mortality has been reduced to the minimum.

Treatment.—Aside from the quarantine, which is necessary for *prophylaxis,* the general treatment of this disease will be, for the most part, symptomatic. Should the *temperature* be very high, the treatment should include cool or tepid sponges, the same as in typhoid. Restriction of the diet to liquids, the employment of cool sponges at stated intervals and the care of the bowels, according to their condition, will all be proper considerations in the management of the case. Normal salt enemas, bathing and the drinking of a sufficient amount of pure water will encourage the elimination by skin, bowels and kidneys, which is so essential to a cure. Should *delirium* appear, and particularly when there are meningeal symptoms, an ice bag over the occiput and cervical region is always indicated. *Fever* is best controlled by the homeopathic remedies which certainly do not contraindicate the febrile sponge. The use of antipyretics should always be discouraged. Treatment for the *throat* may demand hot soda gargles, listerin, guaiac, peroxide of hydrogen (1-3), a carbolic spray (1-60), or the direct application of an astringent like ichtholdin or guaiacolvasogen. *Cold applications,* or often an ice bag, relieve the throat swellings in some cases. *Inunctions* are required as soon as desquamation begins in order to limit the spread of the contagion, and for this purpose carbolized petroleum is as good as anything.

When the bath is given the washings should be rendered innocuous with a germicide, like corrosive sublimate (1-1000). When possible the hair should be cut and the scalp shaved, and as little upholstered furniture as possible should be used in the room. After recovery the formaldehyd lamp should be burned in the room at intervals for a long time, the paper should be removed from the wall and everything which has been in the room should receive prompt disinfection. A weak bichlorid solution should also be used in washing the patient before he is allowed to go free. Much more thorough care should be given to the bed clothing which has been used about the patient. This should be washed and thoroughly boiled at home, and not sent to a laundry. The patient should not be allowed to take cold either during the febrile attack or during convalescence. In case of such mishaps, and particularly during the time of the desquamation, the physician must be on guard against nephritic complications. To be positive about the kidney condition the urine should be frequently examined by an expert in this line.

Serum therapy has not yet been satisfactorily applied in this disease, though there is much commendation for the use of anti-streptococcic serum on the ground of the frequent mixed infection. Most observers claim that this should be given early and a dose of 20 c. c. is recommended and is to be repeated if necessary in severe cases. My experience with scarlatina satisfies me that the mild homeopathic remedy, given according to its true indications, is a sufficient and by all means a much safer remedy.

The *remedies* employed in the early stages are invariably demanded by the symptoms which are paramount. During the invasion period *aconite* most frequently corresponds to the dry surface, the thirst and the restlessness which precede the rash. This remedy, given for a few hours, invariably starts the rash and then its symptoms disappear. *Belladonna* then is the characteristic remedy which next applies in the early stages, as the erythematous blush, the throbbing carotids, the skin sensitiveness, the usual headache and the throat symptoms simulate its symptomatology. *Gelsemium,* with its chilly sensations, cervical gland indurations and a more pronounced myalgia is often suggested by the early symptoms. When either of the above remedies fails to meet the symptoms *veratrum viride* may be demanded to establish the complete appearance of the eruption. The effect is often enhanced by giving the remedies with hot water, and, in some instances, it may be advisable to give the patient a hot bath to bring out the rash. *Rhus tox* will very frequently be demanded in the place of belladonna. It applies, however, more frequently in the atypical cases, and when the rash is darker in hue and more irregular in its development. When it is indicated the systemic infection is pronounced and typhoid features are usually in evidence. *Baptisia* will be useful in prolonged fevers of typhoid character when the intestinal complications are sufficient to produce an additional toxemia and its attending delirium. Such patients generally lie in a stupor much of the time and the infection may involve the glandular tissues as well as the meninges. *Ailanthus glandulosa* is a remedy of unusual value when the symptoms represent an adynamic condition, and general exhaustion is pronounced. Stupor is then a marked feature, while headache, photophobia and mental perversions are usually recognized. The skin is livid, purple or dark in appearance, the rash often being interspersed with blisters. The throat is edematous and dusky red, the cervical and salivary glands are swollen, while the dry and brown tongue, together with the sordes on the teeth, resemble the symptomatology of typhoid fever. The pseudo-diphtheritic membrane is frequently removed by the internal use of this remedy. *Lachesis, naja, crotalus* and *nitric acid* should be compared with ailanthus. *Camphor* should receive recognition when a state of profound prostration exists. It is often used in the early stages when the rash does not appear as it should and the algid condition is pronounced. *Carbolic acid,* in the second decimal potency, is recommended by Cowperthwaite in scarlatina anginosa when there is a tendency to rapid destruction of tissues. *Hydrocyanic acid* is a complement of camphor when edema complicates the rash and petechial eruptions are present. *Apis mellifica* also covers much of the common symptomatology of scarlatina, edema always being a factor and the characteristic throat symptoms are extreme. The peculiar stinging pains are always prominent, meningeal disturbance may be present and the rash may assume a miliary type.

The nervous symptoms of scarlatina may often demand the use of remedies which are usually indicated in the neurasthenic state. Thus *cuprum, zinc, kali phos.* and *asafetida* are frequently demanded while the more excitable conditions will require *hyoscyamus, stramonium, agaricin, cimicifuga, coffea, pulsatilla, tarantula* and similar remedies. *Zinc picricum, strychnia, plumbum* and *argentum nit.* will usually care for the secondary paralyses. Hemorrhagic complications will always suggest *ipecac, arnica, secale, hydrastin hydrochlorate, hamamelis, nitric acid* and *ferrum phos.* The suppurative inflammations and ulcerations are usually corrected by such remedies as *echinacea, hepar sulph., calcarea, silicea, mercurius* and *sulphur.*

Nephritis is always a serious phase during any stage of the disease. This is most often corrected by the proper attention to diet, warmth and rest in bed. *Cantharis* is frequently indicated in this respect for it refers to the burning sensations and the intolerable urging to urinate. This same irritating sensation applies to the eruption which may be vesicular and to the gastro-intestinal canal as well. This remedy may also be demanded without any direct reference to bladder symptoms, as it is often useful when uremia is imminent. *Berberis vulgaris* also relates to a similar vesical disorder with more of a catarrhal complication of the genito-urinary tract. The mucous sediment is also abundant in the urine and pain in the back and limbs is also a decided feature. *Apocynum, terebinth, hellebore, kali bichromicum, merc. corr., benzoic acid, sambucus* and *lithium* may be compared. *Helonias* is frequently indicated in nephritic complications when, in addition to the urinary symptoms and muscular pains, there is more evidence of inflammatory involvement of the kidneys, albumin usually being found in the urine. *Senecio, pulsatilla* and *aletris* may be compared. *Cannabis sativa* has symptoms similar to cantharis but there are usually more symptoms which point to a nervous disturbance. *Phytolacca,* in the low potencies, is efficacious in renal complications when the inflammatory invasion affects the glandular structures and the eruption becomes pustular. This condition would likewise call attention to such remedies as *mercurius, sulphur* and *kali hydriodicum.* The most useful and the most frequently indicated remedy in the nephritic complications which attend scarlet fever is *arsenicum* and this should be continued, when indicated, for a long time. *Arsenicum iodid* is also useful here.

MEASLES.

Measles, or rubeola, may be defined as an acute and highly contagious disease, which is characterized by naso-bronchial catarrh, fever, an extensive mottled eruption and desquamation.

Etiology.—The contagion is not yet known, but there is no doubt that it is due to a microörganism. Streptococci have been found in the secretions of the respiratory tract, while Canon and Pielicki claim the discovery of a special bacillus in the blood, expectoration and the nasal and conjunctival mucus. The infection is intense, the slightest exposure predisposes and immunity is not always gained by one attack. The contagion is conveyed by the nasal and bronchial secretions, the tears and less commonly by fomites; it may possibly be communicated by the breath and a third party; it is not so tenacious as the contagion of scarlet fever and, therefore, articles of furniture are less liable to cause the infection. Children are most frequently affected, but adults are not exempt. After the fourteenth year there is less liability of contracting it and though nursing infants may take it they are less liable. It occurs most commonly in the epidemic form, though sporadic cases are occasionally observed.

Pathology.—No distinctive morbid anatomy has been recognized aside from the nasal and bronchial catarrh and no pathological tracings have been found after death. The complications present the only pathological changes and these relate chiefly to a broncho-pneumonia or a lobar pneumonia in severe cases. The internal organs are engorged with blood and in black measles hemorrhagic extravasations are observed.

Symptoms.—The period of *incubation* is a distinct phase of the symptomatology and this has been determined, by reliable observations, to have a duration of ten days after exposure. The *prodromal stage* which follows lasts usually for three days. This is marked by a slight chilliness or a pronounced chill, which is followed by a slight temperature (102°-104° F.). On the morning of the second day the temperature remits, but rises again toward the end of this stage and continues high until the rash is well established. Simultaneously with the appearance of the prodromal fever inflammation of the mucous membranes is evidenced, and the child appears to have a cold. The conjunctivae, nose, larynx and bronchial tubes are inflamed and coryza, lachrymation, sneezing and a dry, croupy cough, which later becomes moist, are prominent symptoms. The tonsils may be enlarged and congested and the mucous membrane of the pharynx and upper larynx may also be red and edematous. As early as the first day of invasion small bluish-white spots with a narrow red ring—Koplik's spots—may be detected on the mucous membrane of the cheeks, lips and hard palate and are now regarded as diagnostic phenomena in the early stage of the disease. The tongue is usually heavily coated in contrast with the characteristic "strawberry" tongue of scarlatina.

The *eruptive stage* begins at the end of the third, or on the fourth day and is usually preceded by a marked rise in the temperature, which lasts until the fifth or sixth day, when the rash is well established. The face, neck, forehead and trunk are respectively involved and this eruption, together with the flushed skin, which remains from the incubation stage, gives a peculiar mottled appearance to the patient. In twelve to thirty-six hours the eruption is complete. The *character* of the exanthem is typical of the disease. It may be defined as a macular or blotchy eruption with a tendency to imperfect coalescence, sharply contrasting with islands of normal skin which intervene; the boundary between the eruptive area and the normal skin is uneven and crescentic. Minute papules may appear in the center of the macule causing an elevation of the surface somewhat similar to the papular stage of smallpox. Petechiae may be present in severe or malignant cases and a slight swelling of the cervical lymphatic glands is occasionally observed. In two or three days after its appearance the rash begins to subside in the order of its first appearance, a fine "branny" desquamation being noticed. By the seventh or eighth day, in the average case, the rash has entirely disappeared leaving a "blue mottled stain," on the affected parts of the body, the temperature having usually subsided with the full appearance of the eruption. Many of the symptoms, particularly the cough, which characterize the incubation period, may continue for some time after the convalescence.

The *complications* and sequelae are important phases of the disease. The fever may be very high (105°-106° F.) and the dry tongue, delirium and other extreme symptoms may resemble a typhoid invasion. A serious and fatal form known as "black measles" is sometimes a feature of an epidemic and is characterized by subcutaneous extravasations of blood and hemorrhages from the mucous membranes. The most common complications, bronchitis and broncho-pneumonia, may assume serious phases dependent, of course, upon the severity of the attack and the general state of the patient's health. Diphtheria may occasionally exist as a complication and pseudo-membranous inflammations of the larynx and pharynx are not uncommon features. Ophthalmia, otitis, glandular involvement, cancrum oris, meningitis, gastric and intestinal catarrh, arthritis, neuritis and nephritis are all possibilities in the course of this disease. Tuberculosis as a sequela of measles perhaps merits first place in importance. It may develop in the lungs after a bronchitis or broncho-pneumonia or in the bronchial glands, and is influenced in its development by the previous physical condition of the patient.

Diagnosis.—In the midst of an epidemic it will not be a difficult matter to make a diagnosis. In sporadic and irregular forms of the disease it will often tax the most expert ability to make a proper decision. The characteristic incubation of at least ten days, the appearance of the eruption on the fourth day and the fall of the temperature when this is well established are early suggestive signs. Koplik's spots, which appear at least twenty-four hours before the eruption, the catarrhal symptoms, the persistent dry cough and the peculiar mottled eruption are additional symptoms which should always aid in making the diagnosis. The differential diagnosis must consider many similar eruptive diseases. *Drug eruptions* which can be traced to the causative agent are not difficult to differentiate, though there may be much perplexity when a fever attends such conditions. The temperature, however, does not subside with the development of the eruption, the mucous membranes are not affected, the coryza is absent, the central papules are not generally found in the macules and the disturbance usually subsides when the drug is withdrawn. *Variola* offers some perplexing similarities in the early stages and these must be carefully compared with the symptoms of measles. The prodromal fever of variola rises continuously from the first to the third day and drops to the normal on the appearance of the eruption. The gastric symptoms are marked, the pains in the back are prominent, while the cough and catarrhal symptoms are rare and subordinate. The development of the vesicles and the succeeding pustules settles the doubt. Koplik's spots, however, will constitute the best differentiation in favor of measles in the early stage. *Scarlatina* will offer much confusion when it appears in the form of discrete roseola or when it is compared to a confluent form of measles. Scarlet fever shows an eruption as early as the second day, the exanthem appears first upon the neck and chest and has its characteristic scarlet color. The initial vomiting, the severe angina, the lack of febrile defervescence in the prodromal stage, the strawberry tongue and many other scarlatinal symptoms should overcome our doubts. *Typhoid fever* does not usually show the roseola before the middle of the second week, while the relative showing of the pulse, the enlargement of the spleen, the absence of mucous membrane complications and other features of the disease and especially the Widal test will settle the difference. *Typhus fever* has an eruption similar to the asthenic variety of measles. This, however, does not have a prodromal fall in temperature, the fever does not disappear on the seventh or eighth day. The eruption changes from a roseolar form into petechiae, the face is less involved, and the spleen is usually enlarged. The *roseola* of *syphilis* if attended by fever may sometimes be confusing, but the respiratory tract is not implicated, the eruption does not disappear rapidly nor does it give such an extensive mottled appearance. The knowledge of an initial lesion is generally obtained. *Rötheln* will simulate measles more closely than any other disease. The macules are generally smaller and less red, but the other symptoms are very similar. The duration of the prodrome, however, is shorter, the temperature is slight, the general health is not affected much and it runs a shorter course. Sometimes, however, it will be practically impossible to differentiate these two diseases.

Prognosis.—The mortality in measles is not high unless it prevails among a class of children already impoverished by the lack of life's necessities. It may be fatal from its complications or when it follows another infection. Black measles has a high mortality.

Treatment.—The essentials which apply in the treatment of similar infectious diseases should receive strict attention in this disease. The patient must be *kept warm in bed* and due care should be given lest cold is taken and broncho-pneumonia ensue. In this respect physicians frequently make mistakes, for serious consequences have followed when this care was neglected. The proper ventilation is necessary at all times, and yet no prophylactic negligence should be encouraged. I still believe in the darkened room though it is not necessary to have absolute darkness. To obviate this the patient may wear dark glasses or have a shade to protect the eyes. The *diet* should be bland and limited to liquids during the febrile period. *Baths* are not only essential but they minimize the skin irritation to a great extent. Applying carbolized vaseline to the skin after bathing usually gives much relief. The disease is in reality self limited and, therefore, medication is rarely required. When needed it should be given according to the symptomatic indications only. *Aconite, belladonna, gelsemium* and *chamomilla* are frequently demanded by the early febrile symptomatology, while *pulsatilla, euphrasia, sambucus, kali bichromicum* and *arsenicum iodid* will be useful for the inflammation of the mucous membranes and the coryzal discharges. The cough is the most troublesome feature and this will often tax the physician's ability in prescribing. *Drosera, hyoscyamus, cuprum, stramonium, sticta, rumex, causticum, sanguinaria* and *squilla* may be mentioned as frequently indicated remedies. Convalescence will require much caution and care lest the patient get out of bed before all the catarrhal symptoms have disappeared and some of the complications ensue. The treatment of such complications as broncho-pneumonia, pneumonia and tuberculosis is discussed in their respective chapters.

RUBELLA.

Rubella, also termed rötheln, rubeola notha, German measles, etc., has heretofore been regarded as a modified measles or a hybrid scarlet fever, but is now considered as a distinct disease. It is an acute contagious disease without prodromal manifestations and is characterized by a slight fever and "a punctiform rash that fuses into patches less plainly crescentic than those of measles."

Etiology.—Much controversy has existed regarding the true status of this disease. While its distinct entity is now recognized it bears a closer affinity to measles than scarlet fever; its relation to measles is similar to that which exists between varicella and variola. Its contagion is less acute than that of measles or scarlet fever and it runs a far different course than either of these. Its specific organism has not yet been discovered, but it usually occurs epidemically and there is no doubt of its infectious origin. It is probably spread by cutaneous exhalations, the breath, fomites and the clothing, while contagion is a possibility even during the period of convalescence.

Symptoms.—The *incubation* stage—two to three weeks—is twice as long as that of measles but no characteristic symptoms are apparent. The exanthem is usually the first evidence of the disease for, as a rule, the prodromal signs are either so slight that they are not noticed or they are manifest only as the rash appears. The *eruption* is the chief diagnostic evidence and this occurs, like measles and contrary to scarlet fever, first upon the face and then extends to the upper and lower extremities. Red macules appear and disappear rapidly, one part often showing the rash after it has disappeared from another. The character of the eruption is "rose-red, round, usually not serrated and about the size of a lentil, rarely larger or smaller." The spots are not confluent, there is no tendency to hemorrhage, there is no itching and desquamation does not take place—as in the case of measles—at defervescence. Koplik's spots are never found. The *onset* may be characterized by a mild catarrhal condition like measles but this is usually not so pronounced or of long duration. Slight redness and edema of the pharyngeal and palate mucous membrane is an exceptional feature but the measles cough is rarely experienced.

Lymphatic enlargement, particularly the posterior chain, is a more constant characteristic. The tongue presents no peculiar symptoms and the urine is rarely albuminous. The *fever* possesses some diagnostic importance, for, though it is often wanting in the earlier stage, it may have a marked temporary rise 101°-103° F.) just as the eruption appears. Its duration, however, is short and other constitutional symptoms are slight. The *pulse* varies with the temperature and respiration, while vomiting sometimes occurs as the eruption develops. *Convalescence* may take place, in mild cases, in a few days, or the disease may continue, with mild exacerbations, for two or three weeks. A relapse is liable to be more severe than the original attack.

Complications are liable to occur in the form of respiratory disorders like broncho-pneumonia or gastro-intestinal catarrh but these are extremely rare.

Diagnosis.—When we realize that rubella does not occur in connection with an epidemic of measles or scarlet fever it ought to lessen the diagnostic doubt. In the case of *measles* it will be further differentiated by the longer period of incubation, the negative prodromal symptoms and the slight fever. Furthermore we shall distinguish measles by its typical catarrhal symptoms, the dry cough and the character of the rash described under that subject. The presence of Koplik's spots constitutes one of the positive signs of measles. *Scarlet fever* will be more clearly distinguished by its initial vomiting, the early and scarlet rash, the strawberry tongue, the longer duration and the more serious complications. *Urticaria* will be differentiated by the peculiar wheals on the arms and legs, the intense itching, the gastric disturbances and the fact that it is non-contagious.

Prognosis.—A favorable prognosis may be given in the average uncomplicated case. A relapse is usually unfavorable.

Treatment.—Many cases of rubella will require no treatment. A patient kept warm in bed, nourished only with bland food and cared for according to modern bedside principles of nursing will enter a safe convalescence without remedial help in the average case. When symptoms, which demand remedies are present then they may be employed according to the homeopathic principle. *Aconite, belladonna, pulsatilla, chamomilla, bryonia, rhus tox* and *kali bichromicum* are most frequently indicated.

WHOOPING COUGH.

Whooping cough, or pertussis, is an infectious disease characterized by a peculiar coughing paroxysm with a long-drawn inspiration which produces the whooping sound.

Etiology.—The nature of this disease has been speculated upon by numerous theories, none of which have as yet been properly substantiated. The claim that it is simply a slight bronchitis started by an ordinary cold is contradicted by its occurrence only in epidemic form and by the immunity that is usually furnished by one attack. The theory that it is per se a neurosis with a direct lesion of the pneumogastric, phrenic, sympathetic or recurrent laryngeal nerves is also open to much controversy. We may also dismiss the claim that it appears as the result of tracheal or bronchial gland enlargement, which causes pressure upon the pneumogastric filaments. While the infectious theory is probably satisfactorily settled by clinical observations the bacillus has not yet been isolated and no one has as yet explained its action. It is apparent, however, according to Von Leube "that under the influence of the infection an increased irritability of the recurrent laryngeal nerves is brought about and that irritation of certain areas of the respiratory mucous membrane, especially of the interarytenoid region in its lower parts, causes, by mechanical or chemical irritants, the attacks of coughing to appear." Until a positive opinion may be given as to the direct action of a specific organism it is useless to further discuss the etiological theories.

It is thoroughly settled that the disease prevails in the epidemic form though sporadic cases occasionally occur and it would seem that the contagion is not induced simply by a slight contact. The *general health* has much to do with the reception of the infection, and bad ventilation as well as unhygienic surroundings naturally tend to encourage its development. *Measles* seems to have a definite relation to its occurrence, as an epidemic of whooping cough usually follows an epidemic of measles in the same patients. *Children* "between the ages of first and second dentition" are most frequently affected and the epidemics are more common in the spring and winter. *Sex* furnishes no susceptibility, but one attack almost invariably establishes immunity. The *entrance* of the contagion takes place through the respiratory tract.

Pathology.—No specific morbid anatomy, aside from the catarrhal inflammation of the respiratory tract, has been established. This, according to Meyer-Hüni, is especially concentrated upon the posterior surface of the epiglottis, in the interarytenoid and the infraglottic regions. The pathological conditions found after death are those of the complications—bronchitis, broncho-pneumonia, vesicular and interstitial emphysema and occasionally atalectasis.

Symptoms.—The period of *incubation* is somewhat uncertain, though it is possible to recognize a catarrhal stage which is characterized by conjunctivitis, coryza, pharyngitis or the more positive symptoms of a bronchitis with a paroxysmal cough. This may continue for a few days or a few weeks. The *convulsive stage* will be announced by a coughing attack which starts in short paroxysms rapidly succeeding each other and finally terminating by a long-drawn "whistling inspiration" or "whoop." This is succeeded by an expiratory paroxysm the entire attack shaking the whole body and causing the face to become cyanotic. The eyes seem to protrude, the skin is covered with perspiration and the urine or feces may be expelled involuntarily, the cough being relieved only by the expectoration of a tough or frothy mucus which often causes vomiting. The duration of the attacks varies from a few minutes in the early period to nearly half an hour when the disease is well etablished. Mental excitement or even deglutition may excite the paroxysm. The seizures may persist, with periods of relief, for a long time, a convalescence not being completely established until there is an absolute absence of the paroxysms for months. Usually in four to six weeks the attacks appear less frequently, the convulsive feature subsides, the sputum becomes thinner, and in two or three weeks later an absolute decline takes place.

The *complications* which attend will vary in different cases. Epistaxis is common in small children, hemoptysis may follow from violent vomiting or coughing, while more rarely subdural hemorrhage may occur, ulceration of the frenum linguae occasionally takes place, while convulsions may ensue when very young children are affected. Broncho-pneumonia, severe bronchitis, pleurisy, emphysema, pulmonary collapse, pneumothorax, pericarditis, laryngitis and hernia are among the possibilities. The *sequelae* may also include a serious acute nephritis, marked emaciation and many nervous manifestations.

Diagnosis.—It is not to be supposed that any careful observer will mistake a case of pertussis when the characteristic "whoop" has been detected. Inasmuch, however, as this may not always appear in the early stages, it is possible for the best clinicians to misinterpret the bronchial symptoms for asthma, bronchitis or even a severe cold. In some cases the whoop may not be accentuated. *Measles,* with its peculiar cough and the early similarity of coryzal symptoms, may often simulate this disease. The eruption and the appearance of Koplik's spots should help the differentiation. The *cough of influenza* and other respiratory diseases will not have the same paroxysmal distinction.

Prognosis.—While every practician may enumerate many cases which have reached a safe convalescence the severity and fatality of the disease must always be kept in mind. The disease has been regarded by Dolan as ranking third among the fatal infectious diseases in England. It is liable to be more serious in young infants and the state of the general health has much to do with the recovery. The prognosis has a decided relevancy to the complications.

Treatment.—The *prophylactic care* should be as thorough as in other infectious diseases and it is unfortunate that too many physicians are liable to disregard the importance of this. The *general care* should include all the details which are so essential in the treatment of infectious diseases, though it is permissible, during the chronic stage and when the weather is auspicious, to allow the child to be out of doors much of the time. Fresh air day and night is indispensable. A change of climate is also frequently conducive to a more speedy cure. *Local measures* of all kinds have been resorted to with the object of relieving the paroxysmal cough. Gargles and nasal sprays have been favorite methods for the relief of the upper air passages. The peroxide of hydrogen seems to be the favorite agent in this respect. It is to be diluted well and often combined with an equal amount of glycerine for the spray while it may be used in stronger proportion for a gargle. *Inhalations* of iodin, creosote and menthol have also been of slight help. Some favor the use of the Cresoline lamp for a time in the sleeping room, but in my judgment this contaminates the air, which is a better antiseptic agent. The *fumigations* with sulphur and similar agents are apparently serviceable, but these methods give no more relief than such external applications as Roche's epigastric embrocation, which all the grandmothers usually suggest. *Sedatives* are another form of medication which every practician naturally employs for the sake of temporary relief, but, to my mind, these expedients tend to increase the intensity of the paroxysm when they are discontinued. The bromid of soda, 1 to 5 grains every three or four hours, is a favorite prescription with many, and a standard bromoform syrup in teaspoonful doses is likewise popular. Opiates, chlorals, antipyretics, passiflora and creosote preparations have all been experimented with, the hope being entertained that the paroxysm may be suppressed. We have no data to show that this method of procedure is successful. The elixir of heroin and terpin hydrate—one-half to one teaspoonful, every three hours—will probably give as much temporary relief as any of these. Antitussin has been given a trial but no positive results have been demonstrated.

The most decided success will be gained by the use of homeopathic remedies selected according to their symptomatic expression of the pathological irritation. In the catarrhal stage we shall probably get best results from the use of *aconite* when the dry surfaces, thirst and extreme restlessness indicate the first infectious development. *Belladonna* naturally follows when the cerebral congestion is at its height, the cough is irritating and the coryzal symptoms are in evidence. *Ipecac* will be useful when the secretion of mucus causes the throat irritation which induces nausea or when there is an attending gastric disturbance. It pertains also to a bothersome bronchitis and in that respect may be compared to *tartar emetic* with its loose rattling cough and vomiting. *Drosera* is probably our most frequently indicated remedy, as it corresponds closely to the paroxysmal coughing attacks which follow each other in rapid succession until the patient is exhausted. A divergence of opinion exists as to the potency which should be employed when this remedy is demanded. Cowperthwaite claims that it should be administered in two or three drop doses of the tincture and be frequently repeated. My experience confirms the value of the first and second potencies and I have seen cases, which would not respond to the tincture, give prompt results from the administration of the third decimal potency.

The convulsive feature of the disease will often respond to such remedies as *hyoscyamus, stramonium, cuprum metallicum, agaricin* and *gelsemium,* while it may frequently be best, for the sake of temporary help, to give sedative doses of *hydrobromate of hyoscin* (gr. 1/400 to 1/200), *monobromid of camphor* (gr. 1/2 to 1), or an occasional dose of *chloralamid* (grs. 3 to 8). A certain phase of the nervous symptoms will be met by *asafetida, ignatia, moschus, nux moschata,* and *cannabis indica.* We should not lose sight of that class of remedies like *camphor, veratrum alb.* and *arsenicum alb.,* which relates to a depression characterized by the cold surfaces and general adynamia. Likewise we should not overlook the symptomatology which refers to a deeper seated lesion of the nervous system as may be observed under the provings of *zincum phos., zincum pic.* and *argentum nitricum. Naphthalin* in the second or third decimal potency, has been widely used. *Hepar sulphur, kali bromatum, spongia, sambucus* and *sulphur* should not be overlooked when the symptomatology points clearly to any of these remedies. *Corallium rubrum, mephitis, coccus cacti, benzoin, lobelia, turpentine* and the *arsenite of antimony* have been recommended by various practicians. A tablet of corallium rub. 6x and drosera 2x has given good results in my hands.

MUMPS.

Mumps, or epidemic parotitis, is an acute contagious disease which is characterized by inflammation and swelling of the parotid gland. It is occasionally complicated by a similar swelling of the submaxillary gland, the testicles in men and the mammae in women.

Etiology.—The contagion has not been isolated though a bacillus parotidis has been claimed by several investigators. It is highly contagious and children are most frequently predisposed. In rare instances it may occur in early adult life, and boys are afflicted more than girls, and, therefore, metastatic involvement of the testicle is not uncommon. It occurs epidemically with occasional sporadic development. The contagion is presumably established by the body exhalations, the breath and the salivary secretions and is, therefore, sometimes conveyed by fomites and the contact of a third person. It prevails with greatest frequency during the spring and fall months. Immunity is usually gained by one attack, though frequently only one parotid gland is affected by the primary attack and a secondary infection may invade the other gland. The *morbid anatomy* is distinguished only by the swollen and indurated glands.

Symptoms.—The period of *incubation* is usually long, from seven to fourteen days. The *invasion* is marked by lassitude, anorexia, a slight temperature (101°-102° F.), headache and occasional vomiting. Tension is then felt over the parotid gland and this soon begins to swell and involve the surrounding tissues. The swelling being localized just below and in front of the ear. One or both glands may be affected at the same time or in succession. The *tumor* is hard and sensitive to the touch and occasionally it is slightly reddened. The submaxillary and the sublingual glands are frequently implicated. The face is distorted, the jaws are opened with difficulty because of the swelling, and dribbling of saliva takes place on account of the excessive secretion. The *tongue* is coated and there is marked fetor to the breath. In extreme cases we may detect also a slight enlargement of the spleen, while albuminuria may rarely develop.

The *fever* during the inflammatory invasion is rarely high, although it may have exacerbations in which it may reach 103° or 104° F. for a time. The *duration* varies from one to two weeks. The swelling gradually subsides unless abscess formation takes place, which is rare. Orchitis is a most painful complication in men and may be accompanied by epididymitis. Ovaritis, mastitis and vulva-vaginitis are possible complications in women. Otitis media, meningitis, facial paralysis and arthritis are possible but rare complications.

Diagnosis.—This is not difficult when the typical induration takes place. When this is not pronounced a diagnosis is not so easily made. Metastatic swellings and inflammatory invasion of the parotid secondary to injury or operation in this neighborhood will often confuse. The *prognosis* is always favorable as to recovery. In men the orchitis may be prolonged by secondary complications or the lack of proper suspension of the scrotum and atrophy of the testicle may result.

Treatment.—The patient should be kept in bed in case of fever or at least in a warm room until the symptoms have subsided. Otherwise the treatment is entirely symptomatic except in case of orchitis when the testicles should be suspended by a bandage drawn close to the abdomen. The remedies which have been found useful are *aconite, belladonna, baryta iodid, baryta carb., phytolacca* and *mercurius.* For the orchitis *pulsatilla, clematis, spongia* and *hamamelis* should be compared; while for the remaining induration *aurum* and *iodin* should be remembered.

INFLUENZA.

Influenza, otherwise known as la grippe, or catarrhal fever, is an acute contagious disease caused by the bacillus of Pfeiffer. It is characterized by a fever preceded by intense myalgia, catarrhal invasion of the respiratory and intestinal tracts and extreme prostration. It usually appears in the pandemic form and this may be succeeded by various secondary epidemics. It has been traced back to the sixteenth century and we have experienced four or five pandemics in the last century. These started in Russia, rapidly invading that country, spreading then over the greater part of Europe and, finally, in a few months' time, reaching the United States. Fortunately, the duration of these have been short, but its recurrence in milder epidemics has been the source of much suffering and many deaths, particularly during the past ten or fifteen years. The average mortality is not high, but the complications which commonly follow the lack of care, which seems to be the ruling tendency among those afflicted, have caused many unexpected deaths.

Etiology.—The specific cause of influenza is found in the bacillus influenzae discovered by Pfeiffer in 1891. It is a very small dumb-bell shaped organism which stains with dilute carbol-fuchsin solution. The bacilli are found in the sputum, the nasal secretions, the blood and the lung tissues and are very often associated with pneumococci and streptococci. Their migrations or their toxins are liable to establish secondary infections like pneumonia, pleurisy and endocarditis. The *conveyance* of the germ probably takes place through the air and its distribution is universal. It is highly contagious and may be transferred also by fomites. It has the widest prevalence in winter time, though it is not confined to this season. There are no definite predisposing causes unless we accept the predisposition incurred by a depreciation of vitality and the infirmity of old age. While most of the cases occur among those of middle life, the most fatal cases are found among old people. Immunity is not obtained by one attack, though it is claimed that malaria is antagonistic to the existence of influenza. On the other hand the latter disease predisposes toward the occurrence of pneumonia.

Symptoms.—The *incubation period* rarely lasts longer than two or three days and the prodromal phenomena are characterized by muscular and arthritic pains together with an unusual amount of lassitude and debility. The *onset* is usually announced by a chill or a sense of chilliness which is immediately followed by a fever (102° to 104° F.) which generally reaches its acme upon the first or second day. In some instances there may be a morning remission of one or two degrees with an afternoon exacerbation, or again a secondary fever may appear a few days after the primary fever has fallen. During the fever the myalgic pains, the headache, restlessness and prostration are pronounced, while the nervous involvement will be noted by the depression of spirits, insomnia and the occasional presence of delirium. The *pulse* is usually small, feeble and often intermittent; while it is commonly rapid, it frequently shows a bradycardia due to the profound toxemic depression. The temperature rarely remains high after the first stage and if it does we may anticipate a prolonged illness. *Defervescence* may occur by lysis or by crisis.

Several *types* of the disease will be recognized by the symptomatology though it is possible to observe a merging of the symptoms of each type or at least a great diversity in symptoms. The *respiratory type* is of most frequent occurrence and this is at first characterized by conjunctival suffusion and pronounced nasal catarrh. A dry paroxysmal cough due to tracheal irritation, soon succeeds. Expectoration is usually slight and in the typical case there is no evidence of lung involvement. It is just this class of patients, however, in which we are liable to experience lung complications, particularly after any undue exposure. Bronchitis is the most common development, though broncho-pneumonia is often a serious complication inasmuch as a patient is liable to succumb from edema of the lungs. A typical croupous pneumonia may supervene in any stage of influenza, though it is more frequently observed as a complication about the time of convalescence. In the majority of cases it is an atypical pneumonia which gives no sign of a chill or rapid rise of temperature, the symptoms partaking more of the typhoid character with marked cardiac depression. The true influenza-pneumonia is more inclined to assume the catarrhal than the croupous character, and thus many of the physical findings are atypical. The microscopic examination usually reveals a large number of the influenza bacilli, together with the pneumococci and streptococci; thus a marked leukocytosis is discovered in contradistinction to its absence in uncomplicated influenza. Plastic pleurisy is commonly observed, though the purulent form may occur, and abscess of the lung may terminate the complications. Pulmonary tuberculosis may be favored by an attack of influenza.

The *gastro-intestinal* form of influenza is not of so common occurrence as the respiratory form. It is characterized by a similar catarrhal disturbance of the digestive canal and is of frequent occurrence in children. Nausea and vomiting usually precede the febrile attack, and diarrhea, with much abdominal pain, is a troublesome symptom during the disease. Catarrhal jaundice is liable to intervene and appendicitis may be induced by bacillary invasion. A *nervous type* is not an uncommon form of influenza and in this we shall recognize pain as the characteristic symptom. This may occur as severe headache, facial neuralgia, spinal, arthritic or severe myalgic pain. Prostration is usually pronounced, fever is generally absent and nervous excitement is an extreme symptom. The sequelae which succeed this kind of an attack are often serious inasmuch as meningitis, encephalitis, monoplegia, hemiplegia, cord diseases and a perineuritis are occasionally observed. The *cardiac symptoms* may sometimes be the most marked features of the disease, and when this is true sudden heart failure may occur. More commonly a chronic cardiac disorder may remain after the influenza is apparently cured. Other forms of the disease have been termed, according to their symptoms, the *rheumatoid,* the *apyretic* and the *ambulatory type.* When the etiology and pathology of the disease is better known we shall, doubtless, be able to trace many serious consequences to the existence of this apparently innocent disease. Profound and long lasting mental depression may follow influenza.

Diagnosis.—In the case of an epidemic the disease will not be mistaken and the greatest danger will be that more serious diseases will be diagnosed as the "grip." The sudden onset, the shortness of the febrile period, the catarrhal symptoms, the pains and the prostration will be the most distinctive guiding features. Pseudo-influenza or simple catarrhal fever will be difficult to differentiate, but its shorter duration, the milder character and the absence of the above typical signs will serve to make the distinction. *Pneumonia* will be most confusing, though this occurs most often as a later complication, but its marked chill, rapid rise of temperature, distinctive stages, unilateral involvement, absence of nervous symptoms, more typical cough and expectoration, as well as its physical findings will usually leave no doubt in diagnosis. The hurried and labored respiration of pneumonia should also be a valuable guiding symptom. *Typhoid fever* does not begin so suddenly, the intestinal symptoms, the "step-ladder" temperature, the tympanites, rose-spots, splenic enlargement, delirium and sero-reaction, are all so distinctive that a diagnostic error should not exist. *Cerebro-spinal meningitis* may simulate influenza when the latter shows an aggravation of the nervous symptoms, intense headache, convulsions, delirium, muscle stiffness and retraction of the head. A bacteriological examination of the spinal fluid, following lumbar puncture, will be of inestimable differential value. *Pulmonary tuberculosis* of latent character may be excited by an attack of the respiratory form of influenza. In this respect a diagnosis which clearly establishes the character of the disease is not always an easy undertaking. Influenza, however, has its sudden onset, the coryza in the early stages, the myalgia, nervous excitement, exhaustion and shorter duration will also give additional confirmation; while in tuberculosis the temperature record, night sweats and the characteristic cachexia will aid in diagnosis.

Prognosis.—So far as simple uncomplicated influenza is concerned, there is usually slight cause for concern when prompt and careful treatment is instituted. The complications, particularly pneumonia, are always to be feared, the previous state of the patient's health is a marked factor and the sequelae are to be watched with unusual care, inasmuch as many patients are inclined to make light of an attack of influenza.

Treatment.—The patient should remain indoors in all cases and in bed if of moderate severity. *Isolation* is more essential for a safe prophylaxis than is usually considered by the average practician. Disinfection of the catarrhal discharges will also help to overcome the spread of the disease. A *diet* of liquid character will encourage the prompt action of remedies and favor a speedy recovery, while frequent examination of the heart is always demanded. Such complications as pneumonia must always be guarded against, and when this disease appears it will be necessary to meet all the requirements outlined in the treatment of that disease. So far as the *fever* is concerned, it is rarely necessary to use febrile remedies, and antipyretics of the coal-tar group should only be used with caution. Many serious consequences have followed from the indiscriminate use, generally by the patient, of proprietary headache remedies and pain relievers.

The homeopathic remedies serve us best in the treatment of all forms of this disease. *Gelsemium* is probably one of the earliest indicated remedies in our list. This applies to the chill, the myalgia, the headache, the general prostration and the slight fever of the early stages. It also pertains to many of the nervous symptoms and some of the gastric and intestinal disturbances, as well as the neuralgic complications. Its action is always enhanced when it is administered in hot water, and I prefer the tincture in drop doses for adults and smaller doses for children, every two or three hours. We may also administer it by giving the first or second decimal dilutions every half hour or oftener until relief is obtained, when it should be given less frequently. *Bryonia,* without doubt, ranks next to gelsemium and should be given in the lower potencies. Its application is, however, more essential to the respiratory features which appear early in the disease when the lung and pleuritic involvement gives rise to the painful respiration which always attends the first stage of serous inflammation. Its use in the gastric disturbances will be efficient when the dry tongue and mouth, the thirst for large quantities of water and the local sensitiveness to touch is apparent. Its usefulness in relieving the myalgic symptoms will be evinced when the patient complains of aggravation from motion. More than this, the bryonia patient invariably displays a tendency to nervous irritability and constant splitting headache. *Aconite,* in the mild cases, is often the only remedy which is indicated, and this will be called for by the fever, dry surfaces, thirst and great restlessness. *Rhus tox* also refers to extreme restlessness, muscular pains, which are aggravated during damp weather, and a low form of fever with glandular enlargement. Painful swellings of the joints and occipital headache are also representative symptoms when this remedy is indicated. When the headache simulates that of migraine or assumes the neurasthenic pain in the occipito-frontal area, we should think of *onosmodium*. *Eupatorium perfoliatum* is also a valuable remedy for the myalgic symptoms of influenza, as it refers to the bone-breaking pains which attend the fever. Its action upon the gastro-hepatic organs puts it into close relation to *chelidonium, chionanthus, china* and *carduus,* while its control of coryzal symptoms makes it a useful remedy in the early influenza stages.

For the more specific treatment of the early catarrhal symptoms, we shall have use for *belladonna* when the cerebral congestion, the flushed face, throbbing carotids, mental excitement, lachrymation and coryza indicate an extreme congested state of the mucous membranes. *Ambrosia* relates to the watery discharge, while *sambucus nigra* has similar symptoms, though the turgescence of the membrane is more extreme and there is more of a tendency toward a dry, spasmodic cough. *Arsenicum iodid* is influential when the discharge from the nares and mouth are acrid and irritating, *sanguinaria* refers to a profuse, offensive and yellowish discharge, *kali bichromicum* is characterized by a thick, ropy and tough discharge, and *mercurius iodid* covers many of these symptoms.

The relief of pain is usually corrected by the homeopathic remedies. In the extreme cases of the nervous type we may possibly need a sedative of some kind for temporary relief and in obstinate neuralgias the hypodermatic use of morphine may be required in exceptional cases. *Trional* (grs. 15), *chloralamid* (grs. 15-25) and the *hydrobromate of hyoscin* (gr. 1/200-1/150) will usually be of sufficient temporary use for this purpose. The remedies which are so serviceable in nervous diseases will have a sphere of usefulness in these cases. *Zinc phos., cannabis, zinc pic., agaricin, physostigma, actea racemosa, atropin,* and the *arsenate of strychnia* will usually suffice in the second decimal potency. *Elimination* is a factor of great importance in this disease as in all others, and thus sodium salicylate (grs. 10-15) for adults, four times daily is of great benefit.

Diuresis is also enhanced by copious use of pure water.

It is often a good idea to give some mild liver laxative like *chinoidin* (grs. 2 q. d.) *or elaterin* (gr. 1/20-1/16 t. d.). In conjunction with the treatment for elimination, we must curtail the diet to the absolute interdiction of all meats.

The gastric and intestinal conditions are corrected by remedies which are usually serviceable in this respect. Thus *arsenicum alb., nux vomica, hydrastis, cinchona, ipecac* and similar remedies will be demanded, according to their indications. The intestinal disturbances will call for a wide range of remedies, though the diarrheic condition will furnish the most suggestive symptomatology. *Cantharis,* in the early stages, will be one of our most useful remedies. It relates to the violent burning of the intestinal canal, colitis being a characteristic feature of the intestinal disturbance. The burning tenesmus is often accompanied by the discharge of stools resembling the scrapings of the intestine. Thirst is usually violent, food is repugnant and a chilly sensation is distressing. Associated with the intestinal inflammation we invariably find a similar irritation of the genito-urinary tract, giving rise to violent burning and urging to urinate. *Apis, arsenicum* and *mercurius corrosivus* may be compared. *Dioscorea,* in low potency, will usually correct a diarrhea when abdominal pain and gastric neuralgia, with much belching, are the chief characteristics. *Colocynth* has a similar symptomatology, though the pain is more severe and paroxysmal, requiring hard pressure to relieve the spasms. The stools are jelly-like and the dysenteric excitement is always enhanced by the least food or drink. *Cuprum arsenicum* covers a similar class of symptoms, the abdominal pain being even more severe, exhaustion being extreme and convulsions, often of the uremic type, are prominent features. *Plumbum* will be indicated when the abdominal pain is radiating and constipation, with hard stools, alternates with the diarrhea. With this remedy we may always compare *platina, alumina* and *viburnum.* For the involuntary and dark movements which are attended by pain in the limbs, as well as the abdomen, no better remedy than *camphor* is known. This, however, applies in the early stages when the coryza is present and chilliness is marked. *Veratrum album* may be thought of in the same respect, though purging, body coldness and a clammy perspiration are additional symptoms. *Terebinth* will be useful when, with the watery, greenish and fetid stools, abdominal distention is always recognized. In case the remedy is not endured by the stomach it may be given, per enema, 5 to 10 drops of turpentine in egg emulsion. Other remedies will be called for by their peculiar symptomatic demands.

The sequelae of the disease which refer to exhaustion and nervous weakness will require persistent treatment with remedies which tend to correct the anemic condition. *Ferrum phos.* is naturally thought of first in this respect. This may be given in the third decimal potency or in the form of blood-iron preparations, so many of which have already been tested thoroughly by the profession. The *arsenate of strychnia* or the *arsenate of quinin,* in the second and third decimal potency, have been suggested by many practicians of experience. I have frequently employed a tablet of iron (gr. 1), strychnia (gr. 1/60) and arsenic (gr. 1/50), one pill four times daily, with decided success. In place of this it may be well, particularly in younger patients who are anemic, to give a pill containing iron (gr. 1), strychnia (gr. 1/100), phosphorus (gr. 1/100), four times daily. Should the heart show unusual signs of exhaustion, and this is often a feature of convalescence which is too frequently overlooked, the cardiac tonics should be carefully employed until these symptoms disappear. It should always be an object of the treatment to carefully treat the symptoms of convalescence until the patient shows signs of a permanent restoration to the normal health. This will often require a close study of the diet and frequently a temporary change of climate.

LOBAR PNEUMONIA.

The term pneumonia has been employed indiscriminately to cover a variety of lung affections, some of which have no relation to the disease under consideration. Besides the term used here it has also been properly defined as pneumonic fever, croupous pneumonia, fibrinous pneumonia and lung fever. It is an infectious disease, characterized by inflammation of the lung parenchyma, profound toxemia, fever which terminates abruptly by crisis and certain secondary infective processes. It is caused by the pneumococcus lanceolatus of Fraenkel. It is a disease which, even at the present date, has a wider distribution and higher mortality than any other prevailing infection.

Etiology.—The *bacteriology* of pneumonia was not fully established until 1884, when Fraenkel discovered the pneumococcus lanceolatus capsulatus in the sputum of patients suffering with the disease, and this was later confirmed as the causative factor by Weichselbaum. The pneumococci generally appear in pairs, tapering toward the polar ends and are surrounded by an oval capsule. They are usually more virulent in recent lung inflammations, but are not confined to these lesions, as they are widely disseminated and may invade various tissues. They have been discovered in the nasal, buccal and accessory secretions, while the pleurae, pericardium, endocardium, peritoneum, cerebro-spinal meninges and other tissues may develop a resultant inflammation from their presence. Recently the pneumococci have been found in the blood of pneumonia patients, such cases usually running a short and fatal course. Other micro-organisms, the streptococcus, stapylococcus and various bacilli, may be associated with the pneumococcus, constituting a mixed infection, and we may frequently observe the conjoint presence of the pneumococcus and the influenza micro-organism. It is still a question whether pneumonia may be developed by an increased virulence of the organisms which exist in the healthy secretions of the mouth or whether the germs are acquired from outside sources. The epidemic character of the disease would seem to favor the latter theory. The *mode of infection* evidently takes place by inhalation, the bacilli at first localizing in the lung, but later are more widely distributed throughout the body. Secondary infection usually with streptococci, staphylococci and other pathogenic bacteria is of frequent occurrence.

Predisposing influences include the endemic and epidemic outbreaks of the disease. A *previous attack* always renders the patient more susceptible to a second infection, while general depreciation from prolonged chronic disease favors its reception. The patient who takes cold easily is naturally inclined to the disease, while any acute catarrhal inflammation of the respiratory mucous membrane adds to the susceptibility. The winter and spring months show the greatest number of cases, sudden changes of temperature favor pneumonia, as do cold climates rather than hot. *Age* is an etiological factor, inasmuch as young children and those of advanced years are more susceptible. After sixty a fatal termination is always to be feared.

Males are apparently afflicted more than females, traumatism of the thorax may result in pneumonia, while unhygienic surroundings favor its development. *Alcoholics* contract this disease very frequently, and it is this class of patients which is least able to withstand its ravages. Diseases such as nephritis, organic heart disease, carcinoma and diabetes predispose to its development, as well as influenza. Immunity is not acquired by one attack.

Pathology.—The pathology which is distinctive of pneumonia will be confined generally to the lobe of one lung, though it may involve the whole lung. Sometimes we find both lungs similarly involved, and then it is known as double pneumonia. The lower lobe of each lung is most frequently affected, and that of the right lung oftener than the left. Three stages of the inflammatory invasion are recognized, as follows: (1) The stage of congestion or engorgement. (2) The stage of consolidation (red hepatization). (3) The stage of resolution or diffuse suppuration (gray hepatization).

The *stage of congestion* or engorgement ushers in the disease. The capillaries are distended, the alveolar epithelium is swollen and the air cells are filled with debris and red blood corpuscles. On section the color is dark red and the lung tissue becomes heavier, due to the partially filled alveoli; it pits and crepitates on pressure, but it will still float as some air enters the air cells. Blood and frothy serum exude from the cut surface.

Red hepatization defines the next stage. The air vessels are distended with a fibrinous exudate, giving a liver-like appearance to the lung on section. Its tissue is dry and mottled, reddish-brown in color, and the clotted fibrin, red blood corpuscles, pus cells and broken-down epithelium which occlude the air spaces give a granular appearance to the cut surface. Pneumococci, and sometimes streptococci and staphylococci, may be discovered microscopically. Inasmuch as air does not enter into the consolidated part of the lung, its weight is increased and it sinks in water.

A condition of *gray hepatization* represents the third stage. This has been defined as a diffuse suppuration, due to the destruction of the red corpuscles, the increase of leukocytes and the extension of the exudate. The red appearance of the second stage gradually fades into the gray color, which follows the degeneration of the exudate. As the cellular elements disintegrate, resolution follows. If this process is not complicated by further infection or extension of inflammation, absorption of the exudate and resolution takes place. When streptococci are present in large numbers abscesses are liable to occur. These may rupture into the bronchus, pleural sac, or, more rarely, into the pericardium. Usually they are small and multiple, but may coalesce and form one large abscess. The pathologic condition may terminate in induration, and sometimes gangrene may set in, but these are not common occurrences. Infection with the tubercle bacillus may take place during this stage and the disease assume the characteristic features of a developing pulmonary tuberculosis.

The *duration* of the different stages varies in accordance with the degree of inflammatory invasion and the state of the individual health. The first stage generally ends in forty-eight hours, the second stage lasts from four to eight days, and the third stage may last a few days or a few weeks. The transition usually occurs rapidly, except when there is a gradual extension of the inflammatory process by continuity. The extension sometimes occurs by leaps. It is possible for resolution to occur in either the first or second stage, the alveolar contents disappearing by expectoration and absorption. The association of pleurisy with pneumonia is naturally to be expected in every case except those of central pneumonia.

Lesions of other organs are also noted. Endocarditis is a common complication, pericarditis is less frequent, while malignant endocarditis is often accompanied by meningitis. The right heart may contain coagula, which extend into the blood vessels. The heart muscle is pale and flabby, but fatty degeneration rarely takes place. The spleen is congested and slightly enlarged, while the liver may show similar changes. The kidneys commonly show signs of both acute parenchymatous and chronic interstitial inflammation. The gastro-intestinal tract may show a catarrhal condition.

Symptoms.—The *prodromal symptoms* of typical pneumonia are not pronounced. A feeling of malaise, slight thoracic oppression, some chest pain and a vague sense of chilliness and fever may be noticed in some cases for a few hours preceding the onset, which is sudden and severe. A marked rigor is followed soon by fever, which shoots up with great rapidity to

103°-105° F., where it hovers for some time with slight morning remissions. A sudden elevation of temperature later on will indicate the invasion of a new area by the inflammatory process. The skin is dry, the face flushed and the prostration is extreme. Later a typical cold perspiration appears, the sweat often standing out in large beads upon the face. This is more pronounced when there is a rapid decline in the temperature.

As the fever develops there are severe headache and pain in the back and limbs. The shock may be so great at first as to cause a convulsion or sudden collapse. The anxious expression of the face is marked, and soon a short and dry cough with pain in the affected side appears. The respiration is hurried, jerky and shallow. The expectoration, which attends the cough in the early stage, is scanty and rust-colored. Thirst is excessive and vomiting may occur. Herpes on the lips and nose is often a valuable diagnostic sign. The patients lie mostly upon the affected side. The fever generally terminates by crisis in five to ten days in the ordinary case, and all pneumonic symptoms then begin to subside. More rarely it terminates by lysis.

Symptoms in Detail—*Rapid respiration* is characteristic of pneumonia; it is decidedly out of proportion to the pulse rate, the ratio being at first 1 to 2, instead of 1 to 4, as in health. In adults the average respiration will be from 40 to 60 per minute, while in children it may be as high as 60 to 80. The character of the respiration has been described as panting, both inspiration and expiration being short, and the latter is generally attended by a "grunting effort." Dyspnea will be in evidence when the severity of the disease is marked. Rapid breathing will always be aggravated in accordance with the increased area of lung involvement or associate complications like pleurisy, pericarditis, heart failure or general lung edema. The respiratory movement will always be limited on the affected side, but this, to a great extent, may be due to the effort to obtain relief from pain. It is more pronounced in pleurisy.

The *pulse,* as mentioned, is not high except in serious conditions, and if it is higher than 120, apprehension should be felt. The rapid, feeble and irregular pulse always indicates a failing heart, and this may occur at any stage of the disease. The lung consolidation and the toxemic state will both favor this condition. Any previous debility or infection, and particularly alcoholic excess, will cause a heart failure, often attended by marked delirium, early in the disease. We must study the heart in connection with the pulse. Usually the sounds are clear and distinct, the pulmonary second sound being accentuated. When the right ventricle is unable to empty its blood, this sound will become feeble. This will also be recognized by an extension of cardiac dullness to the right, epigastric impulse, a low systolic sound and a shortening of the long pause.

Pain follows shortly after the initial chill, but disappears generally before the crisis. It is stabbing and lancinating in character, and is due to implication of the pleura covering the affected area. It is usually referred to the infra-mammary region, the axilla or scapular region, though it may be reflected into the abdomen, simulating appendicitis. It is aggravated by the respiratory act, but may not be pronounced in the aged.

The *cough* is frequent, short and dry; it is voluntarily repressed at first, since it increases the pain, but as the exudate softens it becomes looser and easier. The *sputum* at first is frothy and mucoid; a little later it has a rusty color, due to the admixture of blood. On account of its viscid and tenacious character it is difficult to expel and adheres to the sides of the receptacle into which it is ejected. Later it becomes more abundant and purulent, and is, therefore, expectorated with greater ease. In the severe type it may resemble prune juice. Under the microscope it shows red blood corpuscles, alveolar epithelium, pus cells, small fibrinous casts and destructive pneumococci.

The *fever* of pneumonia is characterized by a sudden and high elevation following the rigor (104°-105° F.). It generally remains around this high point, with some remission at night or morning, until the crisis is reached. Previous debility or advanced age may favor a lower temperature and sometimes we may observe a pseudo-crisis preceding the termination. The fever breaks by a typical crisis which may occur anywhere from the third to the fourteenth day, being most frequently observed on the seventh or ninth day. A copious perspiration often marks its advent. The period of decline requires only a few hours, but if the termination is by lysis, which is rare, it will last much longer. *Leukocytosis* attends the hyperpyrexia and it may exist for some time after the crisis. The red blood corpuscles and the hemoglobin show slight change during the fever, but there may be a marked decrease during convalescence.

The *cerebral symptoms* will be recognized by a distressing headache and excessive delirium in severe cases. Actual delirium tremens often develops in alcoholics. The rapid increase of the temperature may end in coma and death. In the adynamic form a low, muttering delirium is more characteristic. The symptoms of meningitis are present in many cases. *Digestive disturbances* will be noted by the dry mucous membrane of the mouth and the coating on the tongue, which later disappears, leaving it dry and brown. Anorexia and thirst are in evidence and vomiting may rarely be persistent. Constipation is usual and further digestive errors are evidenced by the slight splenic enlargement. The *urine* shows febrile changes, being scanty and high colored, with an excess of urea and uric acid. The chlorides are usually diminished or absent during the febrile stage and may suddenly reappear about the time of the crisis. Their failure to reappear at this time is said to presage prolonged convalescence or a fatal outcome. Slight albuminuria is common.

Physical Signs.—In the *stage of congestion inspection* detects limited movement of the affected side, more marked when the lower lobes are involved, with rapid and embarrassed respiration. *Palpation* also reveals the limited motion of the affected side and may detect pleural friction. The vocal fremitus is increased also over the area involved. By *percussion* a higher note is heard over the beginning consolidation; a tympanitic note may be heard especially just above the involved area, the so-called Skoda's resonance. *Auscultation* reveals suppressed breathing and what is a very early diagnostic sign, fine crepitant râles at the close of inspiration. On deep breathing the ear may detect the so-called broncho-vesicular sounds.

In the *stage of red hepatization* the signs noted by *inspection* are those of the congestive stage intensified. This is also true of *palpation*. *Percussion* gives a dull note, which may vary from absolute flatness to one slightly tympanitic. *Auscultation* in this stage reveals tubular breathing and often the absence of râles. "It is simply the propagation of the laryngeal and tracheal sounds through the bronchi and the consolidated lung tissue." (Osler.) The vocal resonance is greatly increased. The crepitant râles disappear.

In the *stage of gray hepatization* or *resolution, inspection* and *palpation* disclose little change at first, but later they show a gradual return to normal. On *percussion* the note obtained over the affected area becomes gradually more tympanitic until the normal is established. *Auscultation* in this stage obtains a variety of mucous râles—the finer ones being called the *redux-crepitus.* The consolidated tissue still transmits sound with great intensity, even to a degree which yields bronchophony or egophony. Later the vocal resonance decreases and finally normal respiratory sounds are heard.

Complications—We find that the pneumococcus toxemia may give rise to symptoms which often take the place of the lung consolidation. We also observe that various complications may exist which change the general trend of the disease. Pleurisy is the most frequent complication, as the inflammation extends to the lung surface and often the pleuritic symptoms are more intense than the pneumonic, and then the term *pleuro-pneumonia* is applied. A copious effusion may be present in place of the ordinary plastic form. This accumulation may become purulent, giving rise to empyema. Often the pleural effusions give no definite signs of existence, or the symptoms are so obscure that they are overlooked. In this condition we shall detect a progressive respiratory weakness, increasing dyspnea, a rise in temperature and the local disappearance of râles and breath sounds; while in empyema the septic symptoms are more pronounced.

A *general bronchitis* often makes the symptoms of pneumonia more severe; this co-existing inflammation will cause a higher fever and a more profuse expectoration. The physical findings will reveal a bilateral involvement, more dyspnea and cyanosis as the right heart is oppressed. Moist râles over the bronchi will be detected.

Edema of the lungs is a condition sometimes recognized. It occurs most frequently with those subjects who have been debilitated by some previous disease. Bright's Disease and diabetes are predisposing causes. A copious and frothy expectoration, small bubbling râles, rapidly developing cyanosis, severe dyspnea and a marked tendency to coma are characteristic signs.

Pericarditis is an occasional attendant upon pneumonia, occurring oftener in the pneumonia of children. As it arises from an extension of adjacent pleuritis it will be discovered more commonly in left sided pneumonia. There may be a serous effusion into the pericardium, but the fibrinous exudate is more frequent. Precordial pain and distress, rapid and labored breathing, together with a feeble accelerated pulse, will be distinctive suggestions; when the exudation assumes extreme proportions cyanosis suddenly intervenes. At times the pericarditis may obscure the pneumonic symptoms.

Endocarditis is a common result in pneumonia, the ulcerative or malignant form being most frequently observed. The symptoms and physical findings are not pronounced. Septic manifestations, especially chills, irregular fever and sweats are highly suggestive, and embolism is a most suspicious sign. The association of meningitis has been recently confirmed. A chronic endocarditis may predispose to the ulcerative or the simple form during the course of pneumonia. Cardiac clots have been found in the right heart. Venous thrombosis, embolism of the larger arteries and cerebral embolism are rare occurrences.

Meningitis has been found to exist in complication with the fatal cases of pneumonia frequently attending ulcerative endocarditis. As it involves the meninges on the convexity of the brain it is not so often detected, but there are distinctive meningeal symptoms when the basal membranes are affected. Intense and persistent headache, rigidity, wild delirium followed by stupor, which develops into deep coma, will define the condition. Meningitis usually develops when the pneumonia is at its height, but it may appear at any stage of the disease.

Other complications, such as *arthritis* of the purulent form, is of occasional occurrence during the course of pneumonia, and is found generally in conjunction with meningitis and ulcerative endocarditis. *Peripheral neuritis* is claimed sometimes as a result of pneumonic infection. *Croupous gastritis,* more commonly *croupous colitis, peritonitis, jaundice,* and *acute nephritis* are sequelae which may often appear, if predisposing tendencies favor.

Clinical Varieties.—*Epidemic pneumonia* has been observed in hospital or asylum work and is usually of a malignant type. This is liable to occur where there is any extensive aggregation of people and especially where proper sanitary measures and isolation in case of illness can not be carried out. It may pervade a whole household at one time so that its contagious and epidemic character is established. The pneumonia following epidemic influenza is a good illustration of this feature of the disease.

Typhoid pneumonia in a sense is a misnomer and yet we may find forms of pneumonia in which typhoid symptoms are preëminent, though the typhoid bacillus is not the cause. It is in reality an adynamic pneumonia which takes on the typhoid character as the result of some debilitating condition or disease. A low grade fever, septicemia, diabetes, nephritis and alcoholism are contributing factors. The dry skin, the extreme prostration, the remittent temperature, the delirium and stupor, together with the other general characteristics of typhoid, will settle the diagnosis. Hepatic and splenic hypertrophy will often exist and the recovery is exceedingly slow. A form known as *latent pneumonia* may be recognized in the aged, the distinctive symptoms being obscured. This may be from the fact that other symptoms seem to occlude the cardinal features or the nervous and general manifestations are exaggerated. Another form known as *central pneumonia* is quite characteristic. In this the lesion begins in the center of the lobe and extends so slowly that the typical signs are masked. *Migratory pneumonia,* in which there is inflammatory extension to other parts of the lungs, *malarial pneumonia,* which occurs in patients suffering with malaria, *ether pneumonia,* which follows the administration of ether, and the peculiar feature of pneumonia appearing in children are other varieties which may be differentiated.

Diagnosis.—The abrupt onset, the rapid rise of the fever and the termination by crisis are signs which should rarely fail us. The dyspnea, the chest pains, the peculiar panting breathing, the abnormal relation between the pulse and respiration, the cough and the rusty sputum, together with the physical signs of lobar consolidation, are other distinctive symptoms. The bacteriological examinations demonstrate the cause of these symptoms. *Acute pneumonic phthisis* is often compared with typical pneumonia, but it will invariably furnish a history of previous tubercular involvement; it develops more gradually, the fever is more remittent, no crisis is recognized, the night sweats are excessive, the emaciation is rapid, the sputum is more purulent and copious, contains blood and numerous tubercle bacilli and yellow elastic tissue. The febrile duration is long, the consolidation is most often found in the apex, a cavity is liable to follow this and the general trend of the disease is toward death. *Broncho-pneumonia* will be differentiated by the presence of the streptococcic organism in place of the pneumococcus, and the fact that it is a secondary disease often following such acute infections as measles and whooping cough. It is also characterized by a gradual onset and a fever of irregular type and variable duration, which declines usually by lysis. *Pleurisy with effusion* has not so distinct an onset, the pain is very sharp, stitching and localized, cough is not so marked and yields none of the characteristic rusty sputum, the fever is moderate, of the continuous type and subsides by lysis, prostration is not so pronounced, herpes are not present and leucocytosis is absent or very slight, except in case of empyema.

Prognosis—The mortality from pneumonia is higher than it should be. When we recognize the infectious character of the disease and institute proper treatment, we should be rewarded by a good percentage of recoveries. With our remedies which have undisputed action in lung consolidation, we should be able to obtain a healthy resolution unless unforeseen complications appear. Individual circumstances have much to do with every case. The alcoholic and the debilitated patient do not respond well to any form of treatment. Cardiac failure is the greatest danger; it may come as the result of extensive consolidation, or from the direct effect of the pneumotoxin on the heart. There may also be an underlying endocarditis or a valvular lesion which is aggravated by the pneumonic infection. Advanced age is also an unfavorable factor at all times, the young and healthy being better able to eliminate toxins when prompt and thorough treatment is instituted. Double pneumonia, re-infection and an extension of the consolidation are also grave features. Extreme nervous symptoms, particularly active delirium, must be regarded with alarm.

Treatment.—The treatment of pneumonia has undergone the ups and downs of therapeutic experiment as much, if not more, than that of any other disease. From the day of venesection to the present day of extreme hydrotherapy and serum therapy, the greatest energy has been expended in the effort to find a specific for this disease. Plasters and poultices, blisters and setons, to say nothing of the depression from the burdensome chest coverings, have at last been obliged to give way to the modern idea of the management of an infectious disease. It is unfortunate that the statement of a surgeon to the effect that "pneumonia has no known cure" has reached the attention of the laity through newspaper publicity. Such statements unnecessarily alarm those who may be afflicted and encourage them in the belief that relief comes only from some panacea in the nature of a "germ destroyer" which is too often offered by the advertising quack. Knowledge that pneumonia is a specific disease leads to the belief that only a specific cure may be relied upon. It is equally essential, however, that we treat the patient as well as the disease and for this reason the principle of the homeopathic or "symptom complex" treatment is best. This is true because the method has been tried and proven by a host of practicians of our school for the past century, because it treats more than the cause, because it affiliates a remedy to the individual conditions of a patient, and because it is not antagonistic to any serviceable adjuvant. The treatment should be considered under the two heads of general management and internal medication.

General Management.—One of the great requirements in the treatment of any infectious disease is the maintenance of free elimination. To this end the patient may be given a tepid or cool bath, gentle friction being constantly applied. He should then be placed in a warm bed, well covered and copious warm drinks should be administered. A recumbent or semi-recumbent position should be maintained throughout the course of the disease. A quiet and well aired room, with an average temperature of 60°-65° F., is a necessity. A humid atmosphere is sometimes desired for the relief of the cough and for this purpose we may either use an atomizer or a large pan of water on the stove. It is utterly impossible to care for the pneumonic patient in the midst of noise and filth. Company should not be allowed. The *diet* should be of the simplest character and during the fever it should be strictly liquid. When the fever subsides or there is any marked debility, it may be advisable to resume a more nourishing diet.

Milk is the best food before the crisis, but it should be given in small quantities every two or three hours at first. Meat broths, egg nog, Koumiss and junket may add variety to the diet. In the later stages the solid foods may be carefully resumed, but over feeding should be avoided. Alcohol as a food is a question of consideration in different cases, but the general consensus of therapeutic experience is against its use. The prepared foods are appreciated by some patients. The *bowels* should demand attention from the very first. If the movements are not normal and regular, a saline may be administered, or if this is insufficient, a mild cathartic is permissible. It is my custom to give a high normal salt enema every morning and this will generally relieve the bowels and at the same time help to subdue the fever and aid elimination.

Local Applications.—It seems hardly necessary to say much under this head, inasmuch as the recent consensus of opinion is decidedly unfavorable to local chest applications. In the light of past experience it seems decidedly strange to think of treating pneumonia without a jacket or some counter irritant. The wet and filthy poultice is fortunately a thing of the past, for there can be nothing more disagreeable to a patient than this. The mustard poultice is regarded now only as a necessity for the temporary relief of pain and the plastic dressings which had such a great run for a while are being gradually discarded. To be sure, there may be some exigency which will demand a measure of this kind, for it is still a great question how we may best overcome the extension of inflammation and consolidation. As a specific bacterium is the primary cause, it seems more reasonable to treat the disease from the infectious standpoint; still we must be open to the use of any agent which will reduce consolidation and promote resolution. It has seemed to me beneficial in some obstinate cases, and particularly in residual or latent conditions, to use a plastic dressing, much as I dislike it. In secondary pneumonia I believe this method may well be considered. Ice applied by means of an ice bag over the area of pain in the chest will often give more relief than anything else and it is of decided value in subduing the local inflammation and thus correcting the temperature. This must, however, be well guarded by oiled silk so as to avoid the danger of taking cold from the wet clothing. In the ordinary case a simple flannel covering or a light layer of cotton is the best and the only required measure of chest protection. In my service at Cook County Hospital I have often been amused at the use of the cotton jacket, as it was almost invariably under the patient's arms or about the neck. Yet recovery was not retarded.

Stimulants.—There is a wide difference of opinion as to the value of stimulants in any stage of pneumonia. The weak heart, to be sure, must be supported, but the danger of forcing the heart with stimulants in every pneumonic attack can not be condemned strongly enough. If the principle is applied at all, it should be resorted to only when cardiac failure is imminent, and this will generally appear in the later stages of the disease. *Strychnia* (gr. 1/60 to 1/30), hypodermatically administered, is probably our best agent for this purpose. In severe cases it should be used every three or four hours, but in milder cases a dose night and morning is generally sufficient. A combination tablet containing strychnia 1/60, digitalin 1/100 and nitroglycerin 1/100, has served me well, though it may be wise to exclude the nitroglycerin after a time to avoid the extreme headaches. *Alcohol* as a stimulant is condemned by many, yet it is an absolute necessity with patients who are addicted to its use. When this is taken away from a typical alcoholic, the cardiac depression and delirium are liable to become uncontrollable. During the period of recuperation it is not a bad idea to give the patient a good wine at stated intervals. *Aromatic spirits of ammonia,* in 10 or 15 drop doses, is sometimes tolerated better by the stomach, and answers the purpose of temporary stimulation as well. In other instances it may be best to use spartein sulphate (gr. 1/10) every two or three hours, or some of the more powerful cardiac stimulants like digitalis and strophanthus. *Oxygen* should always be in readiness when other measures fail, though this should not be pushed too far. It is best to administer it by a spray and the cone should never be held firmly over the mouth.

Hydrotherapy.—After the initial bath this must be employed in every case. Whether it may be sufficiently applied as the ordinary fever sponge or the cold pack must be determined in individual cases. In some instances the cold pack or the gradually cooled bath will not only check the temperature, but will act as a nerve sedative as well. It is not natural, however, for every patient to tolerate the shock of cold applications and heat will ordinarily be more pleasing, if not equally beneficial. Whenever a bath is administered gentle friction should be applied.

Special Features of Treatment.—The therapeutic efficiency of *anti-pneumococcus serum* is not yet settled, though the idea is being seriously considered by those who still believe we shall find an efficacious agent in this line. Some of the special ideas of practice are worthy of consideration. One of these is the necessity of *sustaining normal metabolism* by the administration of predigested or easily digested foods at regular intervals. Another considers the *importance* of *active elimination*. This is obtained by the stimulation of the liver and intestinal tract by cathartics and laxatives, by sweats obtained by external heat, by the withdrawal of venous blood when the right heart is overtaxed and by the direct stimulation of the heart by means of strychnia, etc. In line with the principle of elimination the application of remedies for this direct purpose should be considered. Aside from the use of an abundance of water we may first consider the salycilate treatment. This refers to the regular use of sodium salicylate (grs. 10 to 20), every three hours, or it may include potassium acetate (grs. 10 to 20) equally often, and possibly the employment of such remedies as *stigmata maydis* (m. 10 to 20) of the fluid extract every three hours, and one to two dram doses of the aqueous solution of apocynum at the same intervals. For intestinal elimination it may be necessary to administer calomel (grs. 1/10 to 1/4) at hourly intervals until one to three grains are taken, but the salines, such as Carlsbad salts, Rubinat and Hunyadi water or Concentrated Pluto water are generally sufficient for this purpose. One of the best remedies in this line is elaterin—the active principle of elaterium—which may be given in 1/20 to 1/16 grain doses every three or four hours until an active purge is obtained. For the enforced perspiration I know of no better means of inducing sweat than to pack about the patient ears of corn which have been boiled for half an hour and then wrapped in flannel. Hot normal salt enemas will also encourage the sweating process. Cardiac stimulation has been sufficiently referred to in previous paragraphs.

Cleansing of the mouth frequently with mild antiseptic solutions is another feature of adjuvant treatment which is too often overlooked. In case the pneumococci from this source become virulent, they are liable to be a source of re-infection, or at least they may favor the prolongation of the disease. *Leeching* and cupping are still recommended by some experienced practicians inasmuch as this method of treatment has a tendency to relieve right heart engorgement. The *creosote treatment* is highly commended by others and there is no doubt that this is a valuable adjuvant, at least in cases with tuberculous tendencies. I have employed the *guaiacol carbonate* (grs. 10), three or four times daily, with decidedly favorable results.

The use of *oxygen,* as has been referred to, is too frequently delayed for a final effort to sustain a failing heart. Some claim that its use as a regular inhalation throughout the course of the disease is a desideratum of importance. *Intestinal antiseptics* are of highest value as the co-existing intestinal intoxication naturally adds to the toxemia of the pneumonic infection. For this purpose methylene blue—one or two-grain pills four times daily—benzosol (gr. 5) four times daily, and the sulphocarbolates as previously described, are among the standard agents of this character. The high *normal salt enema,* regularly and freely used, aids not alone the intestinal antisepsis, but through its absorption it tends to supplant the loss of chlorids which are no doubt exhausted in their supposedly antiseptic action during the course of the pneumonic fever.

Medicines.—It is our school of practice which has verified the pronounced benefit obtained by the use of internal medication. We have substantially demonstrated that the remedy which relieves the typical symptoms aids a normal resolution better than all the adjuvant measures outlined above. For this reason we look upon the treatment of pneumonia with far less fear than has been generally entertained. In the early stage we have found *aconite, belladonna* or *veratrum viride,* valuable for the reduction of the fever when they are properly indicated. With the exception of *belladonna* it is not best to continue them very long and *veratrum viride* should be watched with greatest care lest cardiac depression ensue. With the aid of these and carefully instituted hydrotherapy the temperature is generally reduced without unfavorable symptoms, so that a crisis occurs after a natural duration of the early symptoms. Great care should be exercised, even in the administration of these febrile remedies. Antipyretics should rarely be employed, as it is possible to cause cardiac depression if we persist in their use. I have become convinced of late that we err too often in this respect. The extensive distribution of the bacilli and their rapid elaboration of toxins account for the sudden onset, the rapid development of symptoms and the termination by crisis. I believe, therefore, that we shall soon learn to let Nature care for the temperature, which seems essential to overcome such profound toxemia. *Ferrum phos.,* in patients debilitated by any other disease, is a particularly useful remedy during the stage of engorgement. For the pain which exists during the early pathologic development I know of no better remedies than *cantharis* and *bryonia.* The former I give in three to five-drop tincture doses every two hours or oftener and my hospital and private experience in this respect has confirmed my theory that it relieves the painful features in the early development of the exudate better than any other known remedy. *Bryonia* serves us well during the second stage.

Phosphorus is the most useful remedy we have; it applies to the period of consolidation when the peculiar "tightness" defines the exudative stage; by some it is claimed that this symptom represents extreme hyperemia instead of exudation. Practically it is most useful during the second stage or the early part of the third. Prostration and oppression of the chest are attendant symptoms inasmuch as there is respiratory embarrassment. It represents also the debilitated and cachectic state, and, therefore, general exhaustion, sweats and hectic signs are indicating requirements. When this is demanded expectoration is difficult and scanty, but pain is not severe. It is my custom to continue this remedy throughout the second and third stages unless the signs of resolution are marked. *Hepar sulph.,* to my mind, naturally follows *phosphorus* when the exudate begins to soften; it is decidedly the remedy of the third stage and refers to a purulent condition. Fever of a low character is generally present when hepar is indicated, and the spasmodic cough with fetid expectoration, perspiration and emaciation would suggest septic conditions.

Silicea will follow hepar beneficially, as it pertains to a longer standing suppurative process.

Tartar emetic has its friends and enemies; it seems to have been quite generally discarded of late. There is no doubt, however, of its usefulness when there is abundant secretion and the lung is inclined to be paretic to such an extent that little sputum is raised; the noisy and abundant râles are quite characteristic; dyspnea, stupor, cyanosis and clammy perspiration are general symptoms. *Ammonium carb.,* or *ammonium mur.,* in low potencies, seem to take the place of *tartar emetic* when similar symptoms exist, and cardiac enfeeblement is troublesome. *Arsenite of antimony* is mentioned by Goodno as a "rival of tartar emetic." Its greatest sphere is in the pneumonia of older people or when the lung paralysis is so pronounced that expectoration is extremely difficult. The symptoms, in addition to a loud rattling in the bronchial tubes, point to a frothy and watery sputum raised with difficulty and a feeble, rapid pulse, with failing circulation.

Sulphur should not be neglected in the third stage, though many careful prescribers have advised the administration of a few doses in the beginning, to be followed by a more characteristic remedy later. Its symptomatology pertains mostly to the later stages of the disease and particularly when the last stages are prolonged. It will help resolution and in many ways it is a complement of phosphorus.

The *iodids* must always be thought of about the middle stages of the disease. No remedies will be more serviceable when the resolution is imperfect and particularly when any latent cause like tuberculosis or glandular complications exist. *Spongia* is distinctly indicated in this respect with children. *Iodin* may give good results in some cases. *Iodid of antimony* is probably more efficient when the sputum is mucopurulent and bronchitis attends. *Iodid of tin, iodid of baryta, iodid of strontium* and *iodid of arsenic* may be considered with profit.

Hyoscyamus, or better, *hyoscin hydrobromate,* holds the record for the nervous and paroxysmal cough when cerebral symptoms are pronounced. *Belladonna* and *stramonium* may be considered in this respect. In a sense *opium* or *codein* may be called into use for the same purpose. *Heroin* and *terpin hydrate* have been used successfully in teaspoonful doses of the elixir, for the same conditions. When the cough is exceedingly irritable and the expectoration is ropy, *kali bichromicum* or *kali bromatum* may be used. *Agaricus* or, better, *agaricin,* may be called for when cardiac excitement, delirium and stomach disturbances are experienced and it will also overcome profuse sweating.

The *typhoid features* of pneumonia may demand other considerations of treatment which would be naturally indicated in typhoid fever. *Baptisia* stands out preëminently among the remedies for this purpose. *Rhus tox, arsenicum, china ars.,* and *phosphoric acid* may have their sphere of usefulness. In fact, we must treat pneumonia in accordance with its clearly defined symptomatology, just as we manage any other disease, and for that reason we may find many indicated remedies.

TUBERCULOSIS

Tuberculosis is an acute or chronic infectious disease caused by the development of the tubercle bacillus within the body and is characterized by nodular lesions or diffuse infiltrations which, in the course of the disease, always undergo secondary changes. Any tissue in the body may be involved by this bacterium. While the disease has been known for a very long time it was, until the early part of the nineteenth century, regarded as a progressive emaciation specifically characterized by a cough, with rapid suppuration of lung tissue presumably caused by taking cold. Heredity was supposed to be the real factor in its development and until recently no physician has been able to convince the public that it is purely an infectious disease in which heredity plays only a predisposing part, except in rare instances. Many investigators recognized the tuberculous new growth as a constant feature in this disease, but it remained for Koch, as late as 1881, to discover the specific cause in the tubercle bacillus. Since that time the disease has been studied with a definite knowledge of its etiology and pathology and the assertion may be safely made that this scientific knowledge is slowly but surely leading to the eradication of this terrible plague.

Etiology—The *bacillus tuberculosis* is "a short, fine rod, often slightly bent or curved and has an average length of nearly half the diameter of a red blood corpuscle." When stained it may present a beaded appearance. It is not motile and its extremities are slightly rounded. It stains with the anilin dyes most rapidly at the body temperature and it is peculiarly characteristic that the dye remains after treatment with acids. Its growth is attained best on blood-serum, glycerin-agar, bouillon and potato. The cultures must be kept at blood heat and growing slowly, do not appear until about the end of the second week. "The colonies form thin, grayish-white, dry, scale-like masses on the surface of the culture medium. Successive inoculations may be made from the cultures and at the end of an indefinite series material from one of them inoculated into a guinea pig will produce tuberculosis." (Osler.) Chemical products, an albuminoid substance and a ptomaine have been isolated during the growth of the bacilli and these poisons, together with the pyogenic organisms are supposed to excite the constitutional features of the disease. Koch's tuberculin is a proteid glycerin extract from the bacilli.

Tuberculosis exists also in cattle, though there are certain points of difference between the human and bovine bacilli so that the disease in man and in animals is not exactly identical. Koch astounded the medical fraternity in 1901 by his statement that man could not be infected by bovine tuberculosis. This claim was forcibly contested and the belief is now held that "in the absence of more positive evidence to the contrary, the bovine tubercle bacillus has a high degree of pathogenic power for man also, and this is especially manifest in the early years of life." On the contrary, there is no doubt, however, that the majority of cases occur in man by the dissemination of germ-laden sputum and infected food. When we take into consideration the innumerable bacilli which exist in the desiccated sputum which fills the air of our congested centers of population it is not necessary to look further for the cause of the disease.

The *number* of the bacilli will vary in the different lesions. In the actively developing tubercles they are very numerous, while in the chronic processes, as observed in lymph-glands and joints, they are few. When a tuberculous focus communicates with a vein or with lymph vessels, the bacilli will be widely disseminated throughout the body. Outside of the body the bacilli are, of course, very numerous and their vitality is extraordinary. Their destruction is not obtained by freezing and they are not easily destroyed by chemical agents, though boiling and exposure to the sun's rays for some time are positive means of destruction. Dried sputum which floats in the air or collects on the contents and walls of a room inhabited by a person with phthisis is a very frequent cause of spreading infection. *Inhalation* is, without doubt, the most common mode of infection and in this way the dried sputum carries the bacilli to the lungs. The claim that infection is established by the exhaled breath of a tuberculous patient is probably untenable other than that the breath conveys minute particles of germ-laden sputum. The pathogenic process commonly develops first in the upper pulmonary area, the small bronchi being the usual site of invasion. The question of contagion has not been clearly settled, but it is reasonable to believe that it may be conveyed by contact. The fact that those who live in close relation with tuberculous patients are inclined to contract the disease is evidence of this possibility.

Swallowing the bacillus may develop tuberculosis as has been instanced in animals and human subjects. *Contaminated milk* may be the means of distributing bacilli and without doubt intestinal and mesenteric tuberculosis is produced in this way. For this reason a tuberculous mother should not nurse her child. Meat may convey it in the same manner when the bacilli are taken into the stomach, though we have not much direct evidence relative to this form of contamination. *Inoculation* may be the means of its transference, as has been clearly demonstrated by experiments upon guinea pigs. The local lesions are occasionally established by inoculation through fissures, excoriations and wounds. This may be illustrated among those whose vocations demand the handling of tuberculous patients and the surgeons who operate upon such patients. Direct *hereditary transmission* was formerly considered a very constant cause of this disease, and, while in rare instances, the fetus in utero has contained the bacillus, as also has the placenta, it is not at all usual for the infection to be so transmitted. Heredity does, however, play a large part as a predisposing factor. The faulty physical development which is a sequence of such an inheritance is either an indication of tuberculosis or a physical factor which will encourage its development at the first infectious opportunity. For this reason so much stress has been laid upon the scrofulous diathesis in children.

Other *predisposing factors* in this disease are worthy of consideration. Nationality is of unusual prominence in its development. It has been found that the negro race and the Indian are peculiarly predisposed in this respect. *Previous infections* are also predispositions and we should always regard an acute infection which involves the lungs as preparing a favorable soil for the development of the bacilli. *Age* does not offer exemption at any time, though children are more inclined to meningeal, mesenteric and glandular, rather than pulmonic tuberculosis, which occurs most often between the ages of twenty and thirty. *Sex* seems to make slight difference, though females are said to be affected most frequently, pregnancy being a factor which, by debilitating the system, favors its contraction, and the indoor life of woman naturally predisposes to lung weakness and the reception of infection. *Environment* has much to do with the etiology as the exposure is certainly enhanced in the city and the conditions of life there are such as depreciate the general health and, therefore, weaken the resisting power. The imperfectly ventilated apartment or workshop, where sunlight is excluded during the daytime, gives the bacilli an opportunity to thrive which does not exist in the country. Among the inmates of our prisons and almshouses who are forbidden the privilege of sufficient exercise and whose quarters are in dark and damp surroundings, the death rate from tuberculosis is a disgrace to modern penal and charitable methods. In fact we may safely say that one of the worst sanitary evils of modern times is the housing of many people together where polluted air is constantly re-inhaled. Soil and climate have some influence favorable to the spread of the disease as humidity and moisture, which prevail in low and poorly drained localities, are conducive to tubercular development. *Occupations* which confine one to an unhealthy locality or a poorly ventilated room or where the atmosphere is dusty, will often debilitate the system beyond the power of resistance, to say nothing of the inflammation which may be set up by such irritants. For this reason we find our largest number of deaths from tuberculosis among the shop workers.

Local conditions may also influence the infection as, for instance, a catarrhal bronchitis, which is chronic or recurrent, establishes a local inflammation favorable to the implanting of the bacillus. Chronic diseases like organic heart disease, arteriosclerosis, nephritis, cirrhosis of the liver, etc., may create a physical debility which encourages the contraction of this infection. Entero-colitis in children predisposes to tabes mesenterica while it frequently follows some of the specific diseases like measles and whooping cough which may establish a bronchial catarrh favorable to its development. Pneumonia also is frequently a forerunner of the disease, especially when resolution is imperfect. Hemoptysis is said to favor it, pleurisy often furnishes a starting point, while congenital or acquired contraction of the pulmonary artery is supposed to predispose to it. Traumatism is a factor to be considered at all times. Pleural and pulmonary tuberculosis often follow from a blow upon the chest; injury to the knee may also establish a tuberculous arthritis; while injury to the head may develop tuberculous meningitis. This is explained by the theory that the bacilli receive a stimulus to their growth by the contusion of the parts and the diminished phagocytic activity following the injury.

General Pathology.—The most frequent seats of tuberculous lesions within the body are the lungs, larynx, intestines, peritoneum, bones, urogenital system and brain, in the order mentioned. In adults the lungs are nearly always attacked; in children the lymphatic glands, intestines, bones and joints are the common seats of invasion by the tubercle bacillus. Any of the viscera may be affected, usually secondarily.

After the bacillus is deposited within the tissues it multiplies and in the course of this process, and as a result of it, there is developed the characteristic *tubercle* which is the essential lesion of tuberculosis. Each tubercle consists of a central focus of *giant cells* (polynuclear), the number of which bears an inverse ratio to the number of bacilli found in and between them. Hektoen claims that these giant cells exert a phagocytic action. In and about this central focus is a zone of epithelioid cells, while external to this are the leukocytes, the whole being held together by a reticulum. This proliferation of fixed tissue elements is due to the specific irritant action of the bacilli. The tubercles vary in diameter (1/2 to 3 mm.), and are translucent at first and when small; later when they are larger and have undergone secondary changes they may have a grayish or yellowish color and reach the size of a pigeon's egg.

Changes in the Tubercle.—The elementary tubercle always undergoes secondary changes, the most common of which are: (1) Fatty degeneration and caseation. (2) Fibroid induration. (3) Calcification. (4) Suppuration. *Fatty degeneration and caseation.*—This begins in the center of the tubercle and is seen first in the giant cells, next in the epithelioid cells, so that finally the nodule is converted into a mass of granular debris within a capsule. This process may take place with varying rapidity, the large, more diffuse nodules breaking down more quickly than the smaller ones. *Fibroid induration.*—This change consists in the fatty degeneration and absorption of the central part of the nodule, while the peripheral cells are gradually replaced by a dense fibrous capsule. This is Nature's effort to repair the damage already done and limit the infection. When it is complete it is the most favorable change that can possibly occur. (Green-Martin.) *Calcification.*—This usually follows caseation and is due to the absorption of the fluid elements and the deposition of lime salts, converting the tubercle into a more or less gritty or stony nodule. It also protects the body against further infection from this tubercle. *Suppuration.*—If the caseous nodules do not undergo fibroid induration or calcification, they eventually break down and suppurate, producing a chronic abscess. The nearer the tubercles lie to the surface of the skin or mucous membranes the more likely are they to suppurate. The "pus" of a tuberculous abscess consists of "fatty granules suspended in fluid, with here and there a fattily degenerated granular leukocyte." (Green-Martin.) It often contains gritty or cheesy masses and is usually thinner and lighter in color than true pus. This suppurative process is very chronic, little subjective evidence being given of its presence until shortly before it bursts when it presents the usual signs of acute abscess. When this stage is reached there is nearly always a periadenitis present also. "The contents of these abscesses are infective and produce general tuberculosis when injected into animals." (Green-Martin.) All tuberculous abscesses are alike in their origin and development, whether found in bone, gland, lung or other viscera or tissues.

TUBERCULOSIS OF THE LYMPH GLANDS.

Scrofula was the word which was used to define this disease previous to its recognition as a tuberculosis of the lymph glands. The presence of the tubercle bacilli within them now account for the specific inflammatory process, while the less virulent character of the infection explains the slow progress and the latent character of the disease. It is most common in children, though it may occur at any age. One fortunate feature of the disease is the fact that the lesion is usually confined to the group of glands first affected. The disease may be studied under two groups, local tuberculous adenitis and general tuberculous adènitis. (1) *In local tuberculous adenitis* we shall find cervical glands most frequently affected and it is a fairly common disease in children of poor heredity or unfavorable environment. The etiological factors in its development may be traced back to an acute or chronic naso-pharyngeal catarrh which favors the advent of the bacilli into the lymph circulation. The tonsils may be the primary source of the infection inasmuch as they freely communicate with the atmosphere and are subject to frequent inflammation. Injuries, skin abrasions and eruptions like eczema may give an opportunity for the bacilli to enter the lymph channels though this is a much less frequent manner of infection.

The *symptoms* at first are not numerous or marked, enlargement of the glands being about the only sign. This may involve the superficial or deep cervical chains or the submaxillary or other glands. At first there is noticed only the presence of small movable nodules; these later enlarge more or less rapidly and as the inflammation extends outside of the gland, periadenitis, with fixation of the glands, is observed. Caseation and suppuration frequently follow; the skin becomes inflamed and finally spontaneous evacuation of the liquefied contents takes place. A sinus, often very chronic in character, usually ensues after rupture and often after surgical evacuation. Resolution may take place without caseation or suppuration.

The *diagnosis* will be made by the symptoms just outlined, the etiology which has been referred to and the positive reactions which may be obtained in the majority of cases by the tuberculin test. A *differential diagnosis* relative to lymphadenoma (Hodgkin's disease), lymphatic leukemia and simple lymphoma must be made. In *Hodgkin's disease* the enlarged glands are less adherent to surrounding tissues, less fixed and have a softer feel; they are not inclined to caseation or suppuration and invade more than one group of glands. In Hodgkin's disease also the growth of the glands is more rapid and is not inclined to affect children so frequently. *Lymphatic leukemia* has a more marked leukocytosis, while simple lymphoma, though affecting single groups of glands, is characterized by a less tender and a less painful enlargement, together with the absence of anemia and constitutional symptoms. *Sarcoma* affects groups of glands, spreads rapidly and from the beginning infiltrates adjacent tissues. The danger of *secondary tuberculosis* following glandular tuberculosis is always possible. The disease usually runs a slow course, but recovery may take place either spontaneously or by early removal of the glands and appropriate treatment to develop the physical health. If care of this kind is not promptly instituted, there may be a diffusion of the bacilli. The *bronchial glands* are often enlarged either independently or in conjunction with lung tuberculosis. Frequently the enlargements are not visible, but they may attain such size as to form recognizable mediastinal tumors. An examination of the glands would reveal the enlargement and the early caseation which is later followed by calcification or abscess formation. Symptoms of pressure may be present and, in the case of an abscess, rupture may take place into the trachea, bronchi or a pulmonary blood vessel, giving rise to cough and expectoration of purulent, bloody and caseous matter containing bacilli. Bronchial catarrh is one of the earliest symptoms in primary tuberculosis of the bronchial glands.

The *mesenteric glands* (tabes mesenterica) may become primarily infected or more frequently affected secondary to intestinal tuberculosis. These cases occur almost invariably in children, the characteristic appearance of such patients being suggested by the anemic condition, the wasted limbs and the protruding abdomen. The local symptoms indicate peritoneal effusion and the enlarged glands or nodules may sometimes be palpated unless the tympany is extreme. Diarrhea is a prominent symptom and fever of an intermittent type attends the progressive cachexia. Death usually occurs as the result of exhaustion, though enteritis or some intercurrent disease may be the cause. Tabes mesenterica may appear in adults as a secondary condition following pulmonary tuberculosis and only rarely as a primary infection. Suppuration is not so common when the mesenteric or retroperitoneal glands are affected as in the case of the cervical. A systemic infection is always to be feared, though spontaneous cures have been reported.

A *general tuberculous adenitis* may, in rare instances, involve all the lymph glands of the body without affecting other organs. It generally starts as a primary tuberculous involvement of the cervical glands. This will be determined by the enlargement and tenderness of the glands with a tendency to suppuration, the protracted fever of intermittent type, the exhaustion, emaciation and its chronic course. Tuberculosis of the lungs or tuberculous meningitis may follow the liberation of bacilli from the local lesions. Some cases may terminate in profound anemia without any definite knowledge of the cause when the glandular enlargement is not marked.

ACUTE TUBERCULOSIS.

This has also been defined as a diffuse general tuberculosis or an acute miliary tuberculosis. It is characterized by the sudden development of miliary tubercles in different parts of the body following the introduction of bacilli into the blood or lymph-channels from some established tuberculous focus. The lesions are found most frequently in the lungs, the pleurae, meninges and peritoneum. The liver, the spleen, the bone marrow, kidneys and other tissues are less commonly involved. Young people, between the ages of twelve and twenty, furnish the great majority of cases, though adult life is not exempt. Though it usually starts from a pre-existing tuberculous focus it may develop secondary to an acute infectious disease like measles and whooping cough in children, or typhoid fever or pneumonia with delayed resolution in adults. *Infection* is commonly established by a "fistulous connection between a local tuberculous focus and a vein, especially the pulmonary vein." The bacilli, entering the blood, may multiply and produce a general infection. A rare form is developed through the rupture from a tuberculous focus directly into the thoracic duct, thus establishing a direct communication with the subclavian vein. Miliary tubercles may exist in certain tissues—the liver, heart and choroid—without giving rise to any characteristic symptomatology. Three clinical varieties of this form of tuberculosis are recognized, the typhoid, pulmonary and the meningeal.

The Typhoid Form.—The *symptoms* of this form of tuberculosis closely resemble those of typhoid fever, together with some distinctive feature of tuberculous invasion. A period of *incubation,* characterized by malaise, headache, debility and some chilliness, usually precedes the onset which, in some instances, may appear suddenly. The fever increases rapidly and usually has a high range (102°-104° F.), sometimes showing the typical evening rise and the morning fall. In other instances it may intermit irregularly or alternate with a continuous temperature for a few days, while in rare instances there may be an afebrile condition. With febrile exacerbations the cheeks present a "circumscribed blush." The *pulse* is small and rapid in proportion to the temperature (100-140). *Respiration* is rapid and labored. A *cough,* with slight expectoration, is usually present in the early stages and this may be profuse and contain bacilli when associated with an old pulmonary tuberculous lesion. *Cyanosis* is sometimes discovered though the characteristic pallor is more frequently observed. *Mental dullness,* or delirium, which often simulates that of typhoid with occasional acute exacerbations, is a typical symptom. *Prostration,* anemia and progressive emaciation are always prominent features. Sweating and herpetic eruptions, with intermitting fever, often simulate malaria. Splenic enlargement, hemorrhage from the bowels and epistaxis are exceptional symptoms. The *digestive* symptoms will be characterized by anorexia, dry tongue, occasional vomiting and excessive thirst. Tubercles may be discovered by means of the ophthalmoscope on the choroid coat of the eye.

A *physical examination* in the early stages does not reveal any prominent chest findings other than a bronchitis, unless there is a primary tuberculosis of the lungs. Lung consolidation and pleurisy generally follow later, while a pericardial friction, peritonitis or meningitis may be detected in many instances. A sputum examination does not usually reveal the bacilli. A blood analysis may possibly reveal them.

The *diagnosis* must differentiate between this disease and typhoid fever. The following characteristic symptoms should help to settle this doubt. There is usually a family history or a local tuberculous lesion somewhere. Epistaxis is not common. The temperature record shows great irregularity. Respiration is rapid and difficult. There are usually no suggestive abdominal symptoms like tympanites, rose spots or splenic enlargement. The Widal reaction is not found. Tubercules may be detected in the choroid coat. Hemorrhage from the bowel and perforative peritonitis are rare. The tuberculin test may demonstrate the existence of the tuberculosis.

The Pulmonary Form.—*Symptoms.* In this form the pulmonary features are pronounced. It succeeds a condition of chronic tuberculosis of the lung while in children it may follow an attack of measles or whooping cough. The symptoms of a diffuse bronchitis are apparent from the first, a cough, marked by slight expectoration, which later becomes muco-purulent, also being present. Hemoptysis is an occasional symptom, but dyspnea is the most striking feature, the respiration often becoming as rapid as forty or sixty per minute. When this becomes extremely labored a peculiar cyanotic pallor is characteristic. The temperature is usually high (103°-105° F.) while the pulse, which is small, feeble and often irregular, ranges from 100 to 150, or even higher in the advanced stages. The physical signs are those of diffuse bronchitis in the early stages or broncho-pneumonia in the more advanced stages. In case of acute emphysema the percussion note may be full and clear in the anterior part of the chest. The râles are sibilant and sonorous, or fine and crepitant at first and later they may be larger and moist in character. High pitched tubular breathing may be recognized at the base of the lung when children are affected. Friction crepitation may be detected in case of tuberculous pleurisy. The *diagnosis* may be difficult, though the antecedent history of a cough or a known family tendency toward tuberculosis will help to confirm the suspicion as to the character of the disease. The tuberculin test will, however, be positive. The bacilli are not usually found in the sputum except in occasional advanced cases, but they should be looked for in the blood. Choroid tubercles may be discovered by the ophthalmoscope. It runs a longer course than general miliary tuberculosis, the only exception being in the case of children. The marked signs of the disease are the disproportionate dyspnea and cyanosis which attend the physical signs of a diffuse bronchitis.

The Meningeal Form.—This is best known as tuberculous meningitis, though it has received many other descriptive terms such as tuberculous leptomeningitis, acute hydrocephalus and basilar meningitis. It may be defined as "an acute inflammation of the pia mater due to an eruption of miliary tubercles on this membrane and on the blood vessels proceeding from it, extending also, at times, to the corresponding membrane of the spinal cord." The primary focus is most frequently located in the bronchial or mesenteric glands, though the bones and the lymphatics are frequently the points from which the bacilli start. In a very few instances the tuberculous development may have its origin in the meninges. It is more common in children than adults, the age of most frequent occurrence being between the second and fifth year.

The *morbid anatomy* will reveal the tubercles most frequently on the pia at the base of the cerebrum and, hence, the term basilar meningitis. In adults the pia of the convexity is more often the seat of involvement, a simple tuberculous deposit often taking the place of the inflammation. A sero-fibrinous or fibrino-purulent exudation is generally found in the region of the base, surrounding the nerves at this point, extending into the Sylvian fissures and occasionally involving the lateral surfaces of the hemispheres. The tubercules appear as small whitish nodules on the membrane particularly on the Sylvian fissures, the number of tubercles apparently having no reference to the amount of exudate. The middle cerebral arteries and those of the anterior and posterior perforated spaces may reveal the tubercles when they are not easily found elsewhere. "The lateral ventricles are dilated and contain a turbid fluid; the ependyma may be softened and the septum lucidum and fornix are usually broken down. The convolutions are often flattened and the sulci obliterated owing to the increased intraventricular pressure. There is a tuberculous endarteritis with the formation of intimal tubercles due to implantation of bacilli from the blood. Proliferation in the adventitia, with invasion of the media and intima, are common, forming nodular circumscribed tubercles. The lumen of the vessel is narrowed and thrombosis may result. The meninges are not alone involved, but the contiguous cerebral substance is more or less edematous and infiltrated with leukocytes so that, anatomically, the condition is in reality a meningo-encephalitis." (Osler.) The spinal meninges in the region of the cervical cord are usually similarly affected and, in rare instances, this alone may characterize the pathology of the disease.

Symptoms of Acute Tuberculosis.—*A prodromal period,* characterized by gastric and intestinal disturbances, headache, photophobia and restlessness usually marks the onset of the disease. Abdominal hyperesthesia, a decreased excretion of urine and emaciation are also features of this period. In some instances the attack may appear abruptly with basal pressure symptoms and much mental excitement. The stage of *cerebral excitement* usually appears insidiously and is characterized by vomiting, severe headache, chills and a slowly developing fever, which may have an evening exacerbation of 102°-103° F. The pulse is slightly accelerated, but may soon become very rapid and somewhat irregular. The skin is dry, the pupils are unequally contracted, while ptosis is also an early sign. Should the onset be sudden, convulsions, delirium and coma may be present. The patient becomes extremely irritable, pain is excruciating and the hydrocephalic cry is a distinctive feature. The headache is increased by the slightest movement, noise or light, the face is pale or alternately suffused, the facial expression gives the appearance of stupor, general hyperesthesia may ensue and the delirium may become very active. Muscular twitchings gradually develop, the patient starts suddenly during sleep, the vomiting persists without apparent cause and constipation is often extreme. Tâche cérèbrale is sometimes in evidence, but the most characteristic symptom is the patient's intolerance of disturbance.

The *second or transitional* stage is characterized by the subsidence of the irritative symptoms, with a tendency to stupor. The patient becomes more quiet, the vomiting is less severe, the headache and pain evidently disappear and the cephalic cry is not so frequent. The abdomen is scaphoid, the head is occasionally retracted, constipation is extreme and the respiration is irregular and sighing in character. The pupils are dilated or irregular and squint may develop as an evidence of tuberculous foci. Convulsions, rigidity of the muscles, tremors and athetoid movements may now appear. A blotchy erythema is an occasional symptom and the tâche cérébrale may be more pronounced than in the former stage.

The *stage of paralysis* is characterized by the increasing coma, and the convulsions or localized spasms of the muscles of the back, neck and limbs. Optic neuritis and paralysis of the ocular muscles are also prominent features of this period. The pupils are dilated, the eyelids are only partially closed and the eyeballs are rolled up. A monoplegia or hemiplegia may also be observed as well as paralysis of the third, fifth and hypoglossal nerves and parts of the pyramidal tract. Diarrhea may now develop, the pulse becomes frequent, small and irregular, the temperature sinks to the subnormal point in the morning and the typhoid state, with low delirium and involuntary passages, may characterize the terminal condition. In case of an impending fatal termination, the pulse will be more erratic, the temperature will rapidly rise to 105°-107° F. and Cheyne-Stokes respiration appears. A leukocytosis may be discovered throughout the course of the disease, the duration of which will cover a fortnight to four or six weeks. Ophthalmic examination will reveal hyperemia of the disk, the choroidal tubercles and the neuritic changes.

Diagnosis.—The diagnosis will depend upon the mode of onset, the characteristic symptoms, the existence of a tuberculous focus elsewhere in the body and the discovery of the tubercle bacilli obtained after lumbar puncture. The tuberculin test will also offer confirmatory evidence. Syphilitic and traumatic meningitis must be carefully compared. *Several types* must be studied. A mild type will be recognized by the slight and late appearance of tetanic rigidity, convulsions and paralysis. A malignant type may appear in adults with the lesion on the convexity. In this the convulsions appear early and are especially severe, death usually occurring shortly after the onset. A chronic type is an occasional form which follows a fixed focal lesion.

Prognosis.—We may always regard this form of tuberculosis as especially serious. The duration of the average case is from one to three weeks, though marked remissions may sometimes suggest an approaching convalescence. Implication of the convexity gives most serious consequences. Recovery in any case is of rare occurrence.

ACUTE PNEUMONIC PHTHISIS.

This form of tuberculosis is also known as acute pulmonary phthisis, pulmonary consumption or galloping consumption. It is a primary or secondary tuberculous infection of the lung characterized by a rapid inflammatory development. It may start from a tuberculous pleurisy or peritonitis or it may come from a chronic tuberculous lesion in the lung which takes on acute exacerbations. It is observed both in childhood and in early adult life, and appears in two forms, the pneumonic and the broncho-pneumonic. The *pneumonic form* displays many of the pathological phases of lobar pneumonia. One lobe of the lung is usually implicated, though it may affect the entire structure. The inflammatory process, in many cases, so closely resembles the ordinary pneumonic pathology that it is often difficult to differentiate. The tubercles are not always found in the acute inflammatory exudate and for this reason the diagnosis may be somewhat doubtful. The characteristic pathology is shown in the presence of small cavities due to the rapid destruction of lung tissue. Tubercles may be found in the hepatized tissue surrounding these cavities and a greater part of the lung may be grayish-white in appearance owing to the aggregation of these tubercles. Wherever caseous degeneration has taken place there is a yellowish-white appearance. In cases of long duration extensive cavity formation may take place or the lung, in rare instances, may be completely consolidated, with areas of caseous degeneration. The bronchial glands are not generally affected. In the *broncho-pneumonic form* the pathology presents many features which are similar to those of broncho-pneumonia. The areas of consolidation at first present a grayish-red appearance which later changes to an opaque-white. The inflammatory product usually undergoes the process of degeneration and the formation of irregular sized cavities which frequently fuse into a large cavity. Miliary tubercles are usually found distributed throughout the lung and on the pleura, the invasion of the latter being recognized by the adhesions and the deposit of lymph. Baumler has defined a form of tuberculosis inhalation pneumonia which depends upon the inhalation of blood into the finer tubes during hemoptysis.

Symptoms.—In the *acute* cases of pneumonic phthisis the *onset* is sudden, the symptoms simulating those of an ordinary "cold." Rigors, pain in the chest, high fever, cough and marked systemic prostration now appear, the same as in pneumonia. Bronchial hemorrhage is liable to be severe and the expectoration is characteristic of the different pneumonic stages, except for the presence of the tubercle bacilli. *Dyspnea* is usually extreme, the patient often suffering with severe attacks of suffocation. The *temperature* rises rapidly to 104° or 105° F., the pulse increasing in proportion. Later the temperature may assume the remittent or hectic type, with night sweats, rapid emaciation and an increase in the muco-purulent expectoration. The *physical signs* in the early stage will be those of lobar pneumonia, one lobe or one lung being involved. The evidence of consolidation, dullness, increased fremitus, feeble or suppressed vesicular murmur, with the later appearance of bronchial breathing, would indicate the presence of an ordinary case of pneumonia. However, at the time of the expected crisis, the disease instead of beginning to diminish, shows marked exacerbations. The temperature rises, the pulse becomes erratic, sweating is more pronounced, and the muco-purulent expectorations are excessive and assume a greenish color. All of these symptoms are accentuated rapidly and soon the physical signs indicate rapid softening. Yellow elastic tissue and tubercle bacilli are easily discovered in the sputum. The patient steadily grows worse until death occurs, or, more rarely, drifts into a state of chronic phthisis.

A *subacute* or broncho-pneumonic form may be recognized by the more gradual onset and the irregularity of many of the symptoms. The chill is not so pronounced, the fever does not show the marked sudden rise, the pulse and respirations, though rapid, are not the same as in the acute form, and the expectoration does not present the regular changes in character which are typical of the active pneumonic form. Hemoptysis may take place during some stage of the pathologic development, but it is not characteristic of any stage. The night sweats are liable to become extreme as the disease progresses and the exhaustion and emaciation simulate that of typhoid fever. The *physical signs* in the early stage resemble those of bronchitis, though later small areas of consolidation appear and these soon give rise to percussion dullness, tubular breathing and subcrepitant râles. Later the physical signs will point to softening with cavity formation. The disease pursues a course of two to eight weeks which is marked by repeated exacerbations and sometimes it enters upon a chronic stage. Recovery may be a rare experience if the patient has a good resistance, though, in case of fusion of the small cavities, it may suddenly assume the characteristics of the acute form and soon terminate in death. In the diagnosis a careful differentiation must always be made between this, acute bronchitis and bronchiectasis.

Acute broncho-pneumonic phthisis in children may follow as the result of an attack of measles, whooping cough or other infections of childhood. This may occur also at the period of dentition when the child makes a slow recovery. Again it may follow a slight fever, the temperature, cough, night sweats and apex consolidation soon giving expression of the real condition. In a majority of cases it would be natural to make a diagnosis of protracted broncho-pneumonia. It is only the developing symptoms, the phthisical temperature characteristics and the persistent cough, with tuberculous expectoration, which really direct the physician's attention to the true nature of the disease. In the later stages it will be possible to confirm this diagnosis by physical examination.

CHRONIC PULMONARY TUBERCULOSIS.

This is more common than the acute form and follows as the result of a septic infection implanted upon a primary tuberculous infection. It has also been referred to as ulcerative tuberculosis because the ulcerative process ultimately ensues.

Pathology.—The character and extent of the morbid process present decided variations. The distribution of the lesions, according to Kingston Flower, has been well described by Osler: "In the upper lobe the primary lesion is not, as a rule, at the extreme apex, but from an inch to an inch and a half below the summit of the lung and nearer to the posterior and external borders. The lesion here tends to spread downward, probably from inhalation of the virus, and this accounts for the frequent circumstance that examinations behind, in the supra-spinous fossa, will give indications of disease before any evidences exist at the apex in front. Anteriorly this initial focus corresponds to a spot just below the center of the clavicle and the direction of extension in front is along the anterior aspect of the upper lobe, along a line running about an inch and a half from the inner ends of the first, second and third interspaces. A second less common site of the primary lesion in the apex corresponds on the chest wall with the first and second interspaces below the outer third of the clavicle. The extension is downward so that the outer part of the upper lobe is chiefly involved. In the middle lobe of the right lung the affection usually follows disease of the upper lobe on the same side. In the involvement of the lower lobe the first secondary infiltration is about an inch to an inch and a half below the posterior extremity of its apex and corresponds on the chest wall to a spot opposite the fifth dorsal spine. This involvement is of the greatest importance clinically as in the great majority of cases, when the physical signs of the disease at the apex are sufficiently definite to allow of the diagnosis of phthisis being made, the lower lobe is already affected. Examination, therefore, should be made carefully on this posterior apex in all suspicious cases. In this situation the lesion spreads downward and laterally along the line of the interlobular septa, a line which is marked by the vertebral border of the scapula, when the hand is placed on the opposite scapula and the elbow raised above the level of the shoulder. Once present in an apex the disease usually extends in time to the opposite upper lobe; but not as a rule until the apex of the lower lobe of the lung first affected has been attacked." The frequency of involvement pertaining to the right or left apex varies in the experience of different authorities, the majority holding that the right apex is most frequently involved first.

The *tuberculous infiltration* starts in the smaller bronchioles and the inside of the alveoli, these parts soon becoming occluded by the inflammatory exudate. At first it is confined to certain lobules, but invades a greater area of lung tissue later. The softening process may now ensue, leading to expectoration when liquefaction takes place, or the morbid products may be absorbed. Associate ulceration may lead to cavity formation or, when caseous degeneration follows, the process of calcification may encapsulate this mass and thus favor recovery. A fibroid induration may supplant the softening and thus localize the infection. The destructive process, together with the increase of connective tissue, may, however, gain the ascendency, causing great impairment of the lung parenchyma, thus favoring death of the parts involved. *Cavities,* varying in size and number, develop as the result of ulceration and tissue necrosis.

There may be new cavities with soft and friable walls which develop in line with the destructive process, often rupturing into the pleural cavities. Old cavities with well defined limiting membranes, the inner surface of which secretes pus, often characterize the pathologic condition. Within these cavities may be found muco-purulent or purulent matter together with tubercle bacilli and other micro-organisms. The contents are often hemorrhagic or of a shreddy character and sometimes gangrene ensues, with a resultant fetid odor to the breath. Trabeculae, which represent the remnants of bronchi and blood vessels, are usually interspersed through the cavities. The greater portion of the apex of a lung may be invaded by a cavity of this character or several cavities may irregularly communicate with each other and with the bronchi. In such instances the lung tissue is obliterated as the result of this excavation until little remains of the normal tissue. In case a large blood vessel suffers from erosion a copious hemorrhage may take place. *Quiescent cavities* are those which display no progressive tendency. These are usually small and single or they may be discovered in the apex in the multiple form surrounded by a dense fibrous tissue. The lining membrane is usually smooth and the process of repair is in evidence. The blood vessels which traverse these are usually closed by an obliterating inflammation, though aneurysms of the arteries in the cavity walls may be observed. A connective tissue development limits the size of the cavity and as a gradual obliteration of the cavity takes place the neighboring lung tissue may undergo fibrosis.

Interstitial pneumonia is a factor associated with the tuberculous degeneration. The inflammation of this character takes place in the alveoli surrounding the tubercles and the consolidation may extend for some distance about these foci and establish a wide area of inflammatory exudate. This may be a simple pneumonic process or it may be a direct result of the action of the tubercle bacilli. The connective tissue increase by its contraction may obliterate the cavities, though the pulmonary tissue may at the same time suffer from compression following this contraction. By this new tissue formation nature endeavors to arrest the progress of the disease.

Disseminated tuberculosis is a form of chronic miliary tuberculosis. The miliary tubercles undergoing fibroid and caseous degeneration are found throughout the lung as well as at the point of the tuberculous focus. They are small grayish-yellow nodules which may at the same time be discovered in other organs of the body. Whatever part of the lung is affected by these may be the seat of tuberculous degeneration, cavity formation or a diffuse inflammatory process. The pleurae are frequently affected by the tuberculous invasion, the fibrinous exudate causing adhesions of the layers while an effusion of serous, purulent or hemorrhagic character is not infrequent. The *bronchial glands,* in acute cases, are swollen, caseous foci are frequently found and calcification often takes place. The *larynx* may also give evidence of tuberculous infiltration and ulceration, while tuberculous endocarditis is recognized in a small percentage of cases and other organs may present evidence of similar lesions.

Symptoms.—The *onset* may either be gradual or abrupt. When of the former character it may be insidious, as it frequently follows other diseases such as bronchitis, influenza, measles or typhoid. The patient may complain of a slight cough with scanty expectoration or a gradual and unexplainable loss of flesh and strength. An afternoon temperature may be inadvertently noticed and in some instances the patient frequently contracts a cold which may give rise to an accentuation of the temperature with occasional night sweats. The *physical signs* are not easily detected except by careful and persistent examination when the apex consolidation and other areas of lung impairment may be revealed. The onset may occur secondary to acute pleurisy with effusion, or to gastro-intestinal disturbances in which anemia and general debility are pronounced, or again it may develop with such laryngeal symptoms as aphonia and hoarseness, with a harsh and racking cough. In some instances the tuberculous development may present its pulmonic phases secondary to some ill-defined and irrelative symptoms such as those of a chronic peritonitis.

The *abrupt onset* gives rise to symptoms which simulate an acute attack of lobar pneumonia. The febrile characteristics, however, do not partake of the rapid and active development of the regular pneumonic stages. The expectoration is more abundant, is constantly blood-stained and the bacilli will usually be found by repeated microscopic examinations of the sputum. The consolidation is apical rather than in the lower or middle lobe of either lung and the attack is prolonged into the chronic stage without termination by crisis. Chills and fever may rarely characterize the acute attack, but hemoptysis is a more frequent initial symptom and this is usually, though not always, followed by the development of active and pronounced pulmonary tuberculosis.

The *symptoms* may be further subdivided according to their local or general character. The *local symptoms* will reveal the deciding features in the evidence of pulmonary tuberculosis. *Pain* will often be a guiding symptom in the early stages and this will be severe, in case of pleuritis or pleuritic adhesions and only moderately severe in other instances. It may be seated at the base, laterally or anteriorly, or it may be located in the interscapular region. It is frequently associated with intercostal neuralgia and its true nature is often overlooked because of the idea that it is rheumatic in origin. *Cough* is the next special feature of importance and is both an early and a late symptom. At first it is dry and "hacking" in character, scarcely attracting serious attention. Later it becomes looser, more paroxysmal and is accompanied by the expectoration of a glairy muco-purulent sputum with grayish-yellow flocculi. *Blood* is often present in the sputum, and in rare instances active hemorrhage may be an early symptom. When cavities develop the sputum is more abundant and assumes a greenish-gray or a greenish-yellow color while mingled with this we may frequently find more or less bronchial secretion. A *fetid odor* is usually detected when the sputum comes from an old cavity or the case is of long standing. In some cases expectoration is not a marked feature of the disease. *Microscopic examination* of the sputum will reveal the presence of pus cells, blood, fat-globules and, more important than all, the tubercle bacilli. The presence of the bacilli gives indisputable evidence that the case is one of chronic tuberculosis, though a negative diagnosis can not be made by the failure to discover them. When they are not found careful examinations should be repeated until this, with the improvement of symptoms, satisfies us that the patient is out of danger. The presence of elastic fibers in the sputum is an important discovery since it is evidence of destruction of lung tissue.

Hemoptysis is another prominent sign of local impairment of lung tissue and appears in from 60 to 80 per cent of cases.

This may be apparent only as a constant bloodstain in the sputum, or it may assume the character of a profuse hemorrhage. There is, of course, a marked difference between the early hemorrhage which is incident to hyperemia and the later hemorrhage which is due to the erosion of a bloodvessel. A *capillary hemorrhage* may also occur in association with cavity formation and this will be recognized by its chocolate color and numular character. While a hemorrhage may give some temporary relief to the pulmonary condition a serious one induces a reaction which is always unfavorable because it is followed by an anemic condition and a state of debility which exhausts the patient. The local pathologic conditions is always aggravated by hemorrhage and thus it is invariably a forerunner of a serious extension of the specific inflammation. *Dyspnea* is also a symptom which is a characteristic feature of the pulmonic changes, though it varies decidedly in different cases. The respirations are usually accelerated—twenty to thirty per minute—dependent, of course, upon the impairment of lung tissue. Pneumonia, pleurisy and other diseases of the lung may increase the respiratory impairment and thus excite this respiratory function.

The *general symptoms* will be characterized, first, by the *fever,* which though showing a peculiar afternoon exacerbation, is nevertheless a constant sign of the stage of tuberculization. The type may vary according to the degree of pulmonary degeneration, a remitting or intermitting character being respectively detected in case of softening and cavity-formation. *Pyrexia* is always indicative of a progressive tissue destruction, while a period of apyrexia is an indication of a limitation of the local lesion during which time the patient usually gives some sign of improvement. In exceptional instances the disease may exist without the coexisting fever. The maximum temperature is usually reached between 2:00 and 6:00 p. m., after which there is a gradual decline until between 4:00 and 5:00 a. m., when the minimum or subnormal point is reached. There are many variations in this temperature curve and a correct knowledge of these is obtained only by the systematic use of the thermometer. The *pulse* in tuberculosis is of much significance. It is increased in frequency, regular, soft and of fairly good volume. It is naturally more rapid at the time of febrile exacerbation, and with this we may often notice capillary and more rarely, venous pulsations. *Night sweats* are essentially a feature of the febrile action, being most pronounced as the temperature subsides toward morning, though sweating may take place at any time when the patient sleeps. During cavity formation the perspiration is greatly increased.

Emaciation is a natural sequence of the prolonged fever, impaired appetite and defective assimilation. To a certain extent it is the result of the infection. It takes place gradually, both the muscular and fatty tissues being involved. Anemia follows as the nutrition is impaired, but there are no marked blood changes. In the early stages the hemoglobin is reduced and when cavity formation takes place a marked leukocytosis may be observed. A progressive debility attends the anemia. Cardiac disturbance will be recognized in proportion to the lung destruction. When the upper lobe of the left lung is retracted the impulse is increased upward which also occurs in case of displacement of the heart toward the right. Functional murmurs may be heard both at the apex and at the pulmonary orifices.

Gastro-intestinal disturbances are common features in pulmonary tuberculosis of long standing. The appetite is impaired, the tongue is usually coated and aphthous ulcers may be found in the oral mucous membrane. Chronic gastritis is a frequent complaint, hyperacidity of the early stages generally changing later to hyperchlorhydria. Vomiting is often a distressing feature, the stomach being unable to retain sufficient food to maintain a nutritive balance. Tuberculous invasion of the pharynx may interfere with deglutition. The intestinal lesions partake of the catarrhal, ulcerative and amyloid character. Constipation is an early symptom, but during the advanced stages a troublesome diarrhea may ensue, due to tuberculous ulceration of the bowel. The *genito-urinary* perversions may be characterized by an albuminuria with the additional presence of casts in some cases indicating a chronic nephritis. Later a tuberculous pyelitis and cystitis may give rise to the pus and blood in the urine. The *cutaneous symptoms* are characterized by the hectic flush which appears with the febrile exacerbations. A moderate cyanosis is often present and pigmentary stains are sometimes detected upon the chest. The *nervous symptoms* will be characterized by the mental state which is always hopeful despite the fatality of the disease. In rare instances a tuberculous meningitis may intervene and focal lesions may follow as the result of tuberculous invasion of the motor brain.

Physical Signs.—*Inspection* will reveal many suggestive signs in marked cases, but this is, of course, of less value in obese patients. The paralytic thorax, in a majority of cases, may be detected by its general appearance; the chest is usually flat, the thorax is long, the intercostal spaces are wide and the ribs slope from the sternum, giving the appearance of an acute angle in the epigastrium. The supra and infraclavicular spaces are depressed, the clavicles and the costal cartilages are prominent, while the lower part of the sternum is also often depressed. The scapulae stand out prominently and a defective expansion of one apex may be apparent early. As the disease advances this impairment may be bilateral or extend to other parts of the lung near the point of the first lesion. The paralytic condition will be more noticeable on forced inspiration. Inspection also offers other confirmatory evidence in the early prolongation of expiration and the appearance suggestive of the tuberculous anemia or cachexia. *Palpation* will, on forced inspiration, confirm the chest immobility when the fingers are placed over the supra or infraclavicular spaces or between the ribs when the lesion is lower down. The apex "lagging" in the incipient stage may be first detected in this way. The tactile fremitus is increased by the growth of the tubercle and the resultant consolidation, though it is always more marked over the right apex. The fremitus is also increased at the base of the lung, though it is diminished in case of pleural thickening or absent in pleural effusion. *Mensuration* is an important adjuvant in connection with palpation for if the difference in chest measurement between inspiration and expiration should be much under two and a half inches it should excite suspicion of tuberculosis.

Percussion is an important feature in the physical examination. The lung resonance is always impaired as consolidation takes place, though this may be masked by the emphysematous condition of the air cells about the lesion. In such instances the sound has been defined as a "tympanitic deadened sound." Dullness below the clavicle, or sometimes above it, is one of the earliest signs. The two sides of the chest should be compared, both during the completed inspiration and of expiration. In the incipient stage the percussion note over the consolidated area is usually higher. The supraspinous fossa and the interscapular area may also reveal dullness. In recent consolidation from caseous pneumonia the percussion note often has a tubular or tympanitic quality." As a larger area of lung tissue becomes involved the deadened sound is increased until absolute dullness is present. In the case of cavities the percussion note will be modified by the character of the walls, the amount of secretion within and the position of the cavities. While the resonance is impaired by the consolidation about the cavity, the character of the note over that area is somewhat tympanitic. The closure of a connecting bronchus, the filling of the cavity, and a thickening of the pleura will greatly impair the resonance. "Small cavities in the upper area will yield a decided tympanitic note while a large cavity at the base will not." When the walls of the cavity are smooth, a metallic sound will be noted. Wintrich called attention to the louder note and the higher pitch when the mouth is opened wide and the lowered pitch when the mouth is closed. Garhardt noted the change of pitch in the tympanitic note when the position is changed in the case of a cavity communicating freely with a bronchus. The cracked-pot sound is elicited over large thin-walled cavities.

Auscultation gives the most marked diagnostic findings in the chest examination. At first the breath sounds are feeble on account of the limitation of air entering the tubes and air vesicles of the involved area. On the other hand during the early stages the sounds may be sharpened by the narrowing of the smaller bronchi. Deep breathing will often reveal a jerking or wavy inspiration which is termed the cog-wheel rhythm." The corresponding sides of the chest should be compared both during quiet and forced breathing. The prolongation of the expiratory murmur is very significant of early tuberculous involvement. As the consolidation extends the inspiratory sound becomes harsher, "whiffing and bronchial." When the consolidation is complete bronchial breathing is audible and crepitant or subcrepitant râles are detected. A clicking râle, though rarely heard, is a positive diagnostic sign. Whistling râles are often heard with a long expiration during the early stages. These become moist and bubbling as liquefaction takes place and they are heard both on inspiration and expiration. When the râles are scanty they are detected only on inspiration, though the sounds are accentuated by coughing. The normal breath sounds may be entirely obscured when the crepitant and subcrepitant râles are numerous, though a fit of coughing may temporarily restore them. Vocal resonance is generally increased during all stages of consolidation and bronchophony and pectoriloquy may be heard over the consolidated area and over a cavity. Pleuritic friction sounds, in case of an attending pleurisy, may be heard at any stage, though most frequently during the early period, and are often accompanied with râles. Pleuropericardial friction is heard when the lung area over the heart is affected and, in such conditions clicking râles may be developed by the heart's systole, especially during inspiration.

The *signs of cavity,* as enumerated by Osler, may be recapitulated as follows: "When there is not much thickening of the pleura or condensation of the surrounding lung-tissue, the percussion note may be full and clear, resembling the normal note. More commonly there is defective resonance or a tympanitic quality which may at times be purely amphoric. The cracked-pot sound is only obtainable over tolerably large cavities with thin walls. It is best elicited by a firm, quick stroke, the patient at the time having the mouth open. In those rare instances of almost total excavation of one lung the percussion note may be amphoric in quality. On auscultation the so-called cavernous sounds are heard: (1) Various grades of modified breathing—blowing or tubular, cavernous or amphoric. There may be a curiously sharp, hissing sound, as if the air was passing from a narrow opening into a wide space. In very large cavities both inspiration and expiration may be typically amphoric. (2) There are coarse bubbling râles which have a resonant quality and, on coughing, may have a metallic or ringing character. On coughing they are often loud and gurgling. In very large, thin-walled cavities, and, more rarely, in medium sized cavities, surrounded by recent consolidations, the râles may have a distinctly amphoric echo simulating those of pneumothorax. There are dry cavities in which no râles are heard. (3) The vocal resonance is greatly intensified and whispered pectoriloquy is clearly heard. In large apical cavities the heart-sounds are well heard, and occasionally there may be an intense systolic murmur, probably always transmitted to, and not produced, as has been supposed, in the cavity itself. In large excavations of the left apex the heart impulse may cause gurgling sounds or clicks synchronous with the systole. They may even be loud enough to be heard at a little distance from the chest wall. A large cavity with smooth walls and thin fluid contents may give the succussion sound when the trunk is abruptly shaken (Walshe), and even the coin sound may be obtained. Pseudo-cavernous signs may be caused by an area of consolidation near a large bronchus. The condition may be most deceptive—the high-pitched or tympanitic percussion note, the tubular or cavernous breathing, and the resonant râles simulate closely those of cavity."

The *complications* of chronic tuberculosis are also of comparative importance. The *larynx* is frequently affected, manifested first by huskiness of the voice, aphonia, dysphagia and a wheezing cough. When the epiglottis is affected and the ulceration extends to the arytenoid cartilages, the pain during deglutition will be intense and the coughing spells may cause the regurgitation of food through the nostrils. The *trachea* and the *bronchi* may be similarly affected by the invasion of the tuberculous inflammation. *Pneumonia* is a frequent complication which, by its active symptoms, or delayed resolution, may seriously augment the disease. Emphysema is frequently observed in the uninvolved parts of the lung. *Gangrene,* as the result of sloughing of the cavity walls and rarely of the lung tissue, is an occasional sequence. *Pleurisy,* with effusion in the early stages or the dry form with adhesions may complicate and prolong the disease. *Chronic nephritis* is liable to be an associate disease in cases of prolonged tuberculosis. *Chronic endocarditis,* particularly affecting the tricuspid valves, is an occasional sequence of the disease.

Diagnosis.—In advanced cases with marked physical signs and the finding of bacilli in the sputum, a diagnosis is easily established. The greatest difficulty is experienced in the incipient stages when neither the physical findings nor the microscopic examination are confirmatory. In such instances repeated physical examinations and a careful watching of the patient are essential until the bacilli are detected in the sputum. The presence of elastic fibers will give positive information of the destruction of lung tissue and these may sometimes be found before the physical signs are clearly elicited. Heredity and predisposing diseases should carry weight when the disease is suspected. Hemoptysis is always suggestive but the other causes of this should be carefully excluded. A localized and persistent induration in one apex is a most valuable sign. With this we must look for a defective expansion, deadening of the percussion-note, impairment of the normal vesicular murmur and a prolonged expiration. The attendant symptoms of cough, fever, progressive emaciation, and expectoration will help to confirm the diagnosis. The *tuberculin test* is of much value. A positive reaction will be revealed by an elevation of temperature (at least 101° F.) in twenty-four or not later than forty-eight hours after its injection. The differential diagnosis must include the careful consideration of *bronchial catarrh.* In this the temperature record will not show the afternoon exacerbation, localized dullness does not exist in the apex and moist râles, varying in intensity, are heard on both sides and often at the base. The râles clear up after treatment, prolonged expiration is not observed and the gradual loss of flesh and strength are not pronounced. The microscopic examination of the sputum, particularly in advanced cases of phthisis, will settle the doubt. Bronchiectasis should offer no confusion after a careful study of its symptomatology.

FIBROID PHTHISIS.

This is a form of pulmonary tuberculosis in which there is an overgrowth or connective tissue which results in a contraction of the invaded lung tissue. It may follow pulmonary tuberculosis or it may be a primary condition which precedes the pulmonary lesion. The pathologic development is usually confined to one apex, though it may start in a lower lobe and gradually invade the entire lung, thus resembling cirrhosis of the lung. It is unilateral and as the cirrhotic development takes place it is attended by dilatation of the bronchi. The lung tissue is hard and the alveoli are obliterated. On section it is smooth and dry, though caseous degeneration may be occasionally observed. The bacilli may sometimes be found in the walls of an advanced cavity at the apex or the tubercles may be found in the apex of the other lung or in the bronchial glands. The pleura is thickened, its layers often being adherent. The right ventricle and occasionally the whole heart is hypertrophied while the uninvolved portion of the lung tissue becomes emphysematous.

Symptoms.—The symptoms are chiefly characterized by their *insidious onset,* which is usually emphasized by an obstinate and paroxysmal cough with purulent expectoration. *Dyspnea* is noticed upon exertion, but the fever is slight. Emaciation is not pronounced. In case of bronchiectasis the sputum will be decidedly fetid. The *physical signs* are characterized by the sunken chest and the lowered shoulder on the affected side. The heart is usually displaced and murmurs are common. *Dullness* is usually detected over the affected side and the tactile fremitus is absent. Cavernous sounds may be heard at the apex and bronchial breathing at the base. The disease assumes the chronic form, the other lung may be involved later and serious hemoptysis may take place at any time. *Dropsy* may follow as the result of the cardiac weakness while amyloid degeneration of the liver, spleen and intestines is not exceptional. The disease is liable to pursue a long course and the patient may die of some intercurrent trouble.

Course and Duration of Pulmonary Tuberculosis.—No definite rule can be ascribed to the course and duration of this treacherous disease. Much depends upon the early treatment, the environment and the habits of the patient. The great increase in the percentage of cures recently attained is the result of the outdoor treatment and the systematic study of each patient's needs. In this respect our therapeutic ideas have changed decidedly in the last few years and for that reason it is not reasonable to pronounce this an incurable disease. The average duration is put down as about three years, though wide variations occur. Death most frequently occurs by gradual asthenia, the patient retaining hope until the end. It may be induced by asphyxia in acute miliary tuberculosis and in acute pneumonic phthisis. Again it may occur by syncope as the result of overexertion. Hemorrhage is a cause of death when there is erosion of a large bloodvessel or the rupture of an aneurysm in the lung cavity. Coma may also occur as the result of meningitis or uremia and the patient may die in the midst of convulsions. Intercurrent diseases like bronchitis, pneumonia, pleurisy, amyloid degenerations or Bright's disease may also terminate chronic tuberculosis.

Concurrent Infections are always concerned in the fatalities of the disease, as most cases will reveal the presence of other organisms. The micrococcus lanceolatus, the streptococcus pyogenes and the staphylococcus aureus are frequently concerned in the mixed infection. Advanced age favors early death from this disease. Old people are inclined to contract tuberculosis, though the fatality is liable to arise from an exacerbation of the latent disease. In children pulmonary tuberculosis may be a secondary development from glandular involvement or it may be excited by the infectious diseases so peculiar to childhood.

TUBERCULOSIS OF THE ALIMENTARY CANAL

Tuberculosis of the *lips* is not of common occurrence. It may appear primarily as an ulcer or be secondary to laryngeal or pulmonary tuberculosis. The ulcer is sensitive and may simulate a chancre or epithelioma. The presence of tubercle bacilli or the reaction from tuberculin will determine the diagnosis. The *tongue* may reveal an aggregation of small granules on its dorsum or sides which are tuberculous. The disease spreads slowly and as it forms an ulcer it is frequently mistaken for specific disease or cancer. Microscopical examination and the tuberculin test will settle the diagnosis though it may be differentiated from a syphilitic ulcer by its failure to improve under the administration of the iodids. The hard and soft *palate* may be affected by the extension of tuberculosis from neighboring parts. The *tonsils* are often infiltrated with miliary tubercles resulting in hypertrophy with occasional foci of caseous degeneration. The tonsil is frequently the atrium through which glandular and pulmonary tuberculosis develops.

The *pharynx* may be the seat, on its posterior wall, of miliary granules or an ulcerative tuberculous pharyngitis may exist as the result of an extension of the infection from the larynx. Adenoids in the naso-pharynx may also be tuberculous. The *esophagus* is rarely affected by tuberculosis and the same is true of the *stomach,* though ulceration of this character may, in rare instances, give rise to the hematemesis and occasionally to perforation.

The *intestines* are frequently the seat of this disease and the lesions may be primary in the mucous membrane or secondary from tuberculosis of the lungs or peritoneum. The *primary* lesion occurs most frequently in children, though it may be then associated with mesenteric gland enlargement or with peritonitis. When the disease is primary the bacilli are probably swallowed by the child and it may take place, though rarely, in adults. The *secondary variety* is a sequence most frequently of pulmonary tuberculosis and less often of tuberculous peritonitis, the lesion being located in the upper part of the colon, the upper part of the ileum and the cecum. The rectum is occasionally involved secondarily and, more rarely, primarily. The morbid process starts in the solitary and agminated glands or on the surface of, or within, the mucosa. The caseation and necrosis lead to ulceration. "The ulcer is (a) irregular, rarely ovoid or in the long axis, more frequently girdling the bowel; (b) the edges and base are infiltrated, often caseous; (c) the submucosa and muscularis are usually involved; and (d) on the serosa may be seen colonies of young tubercles or a well marked tuberculous lymphangitis." (Osler.) Perforation of the bowel may take place and in that event peritonitis will follow. In chronic cases cicatrization may occur and cause stenosis of the bowel.

The *symptoms* are usually characterized by an irregular diarrhea which increases in frequency and persistently resists treatment. *Moderate fever,* colicky pains, and, in some instances, hemorrhage are features of the initial symptoms. It is usually regarded as a catarrhal colitis until the emaciation, temperature curve, night sweats and lung complications direct our attention to the true cause. In case of constipation or irregular diarrhea, together with tenderness in the right iliac fossa, it may be mistaken for appendicitis when the cecum is involved. In children the disease will simulate a protracted catarrhal enteritis, the diarrhea, the colicky pains and the presence of blood and mucus in the stools often causing the error in diagnosis. In the majority of cases we mistake the disease until the irregular fever develops and emaciation becomes noticeable. Aside from the symptoms the diagnosis may sometimes be made by the examinations of the feces or the mucus which may be passed from the rectum.

TUBERCULOSIS OF VARIOUS TISSUES.

Tuberculosis of the Serous Membranes.—The serous membranes are affected by the tuberculous infection secondary to pulmonary and intestinal tuberculosis while primary tuberculosis of these parts is also a possibility. The morbid process will simulate the ordinary inflammatory invasion of the membranes plus the nodular tubercles. These are generally found over circumscribed areas though they may be diffuse when numerous. The effusion is sero-fibrinous, though it may become purulent and rarely hemorrhagic. The acute form follows from inoculation of the peritoneum or pleura by foci in the bronchial, tracheal or mediastinal glands or from the Fallopian tubes in females. The chronic form follows the acute attack or it may develop by direct extension from some adjacent organ.

Tuberculous Pleuritis.—This appears in the acute and chronic form, the first being ushered in by chills or rigors and attended by the sharp pains, fever and cough which usually are present in pleurisy. The chronic form is more common, being frequently secondary to pulmonary tuberculosis. Adhesions may form as the result of the plastic exudate, or a form of sero-fibrinous pleurisy may ensue. The effusion may be purulent or hemorrhagic, and in case of perforation a pneumothorax may develop. The *symptoms* in this form are of slow development, the physical signs which are characteristic of pleuritic effusion being most pronounced. The gradual development of pulmonary involvement with its physical findings is a secondary condition which soon develops.

Tuberculosis of the Pericardium.—This affection may occur in the acute or chronic form though it is not of unusual prominence. The effusions are either excessive or more commonly slight thickening of the layers and tuberculous deposits are distinctive pathologic features. The *acute form* is of more common occurrence among children, being usually a secondary affection following tuberculosis of the bronchial or mediastinal glands, or a primary pulmonary tuberculosis. The symptoms are similar to those of acute pericarditis which have already been described. The *chronic form* also succeeds the tuberculous infection of the bronchial or mediastinal glands or it may be a part of a general invasion of serous membranes by the tuberculous process. It is likewise referred to as an extension from a tuberculosis of the spine and, in rare instances, it is a primary affection. It may be recognized without effusion, the adhesions leading to hypertrophic cardiac disorders, or, more rarely, it may be recognized by an excessive effusion. In the larger number of cases the symptoms will resemble those of adherent pericardium, the dullness, however, often extending higher up. In conditions of effusion the physical signs are similar to those of cardiac dilatation.

Tuberculosis of the Peritoneum.—This form of tuberculosis is usually a secondary process produced by the extension of the tuberculous inflammation from adjoining tissues. The intestines are most frequently the primary seat of the disease, the bacilli being conveyed to the peritoneum later. The pleurae are also liable to contribute the secondary infection to the peritoneum, while in females it may have its origin in the generative tract. In both sexes the appendix is rarely the primary source of the disease. Age is a factor of importance in the etiology, most of the cases occurring in the early period of life, young children furnishing a large proportion of the cases. Sex is a predisposing influence, the greater majority of cases appearing in the female inasmuch as the Fallopian tubes are a common seat of the tuberculous lesions.

The disease assumes both acute and chronic characteristics, the former being recognized by peritonitis in case of perforation into the peritoneal sac or in conditions of acute miliary tuberculosis. It may take on the *ascitic form* when the exudate is purulent or sero-purulent; again it may assume the *ulcerative form* when the tubercles undergo ulceration following caseous degeneration; and still further a *fibrous form* is recognized by the adhering peritoneal surfaces. In the latter condition the exudate is slight, the tubercles, if numerous, being widely disseminated or localized in small areas.

The *symptoms* of the acute stage appear abruptly, the constitutional disturbance being announced by violent abdominal pain, vomiting and occasional diarrhea. The temperature at first is usually high (103°-104° F.), while again it may not show any decided exacerbation. The peritoneal effusion, not usually excessive, appears very soon, and, when pus is present, it will be announced by sweats and the characteristic temperature. When there is no ascites palpation may reveal the nodular masses and auscultation, at times, may detect a friction sound. Tympanites may be an associate symptom. Anemia, emaciation and the typhoid state are secondary symptoms. The *chronic form* is characterized by slight fever, anemia, pains and abdominal distention, though many times the symptoms are so latent that they are overlooked. The fibrous form gives indistinct symptoms. With the anemia and emaciation there is often only slight fever, with the usual local symptoms of pain and distention.

The *physical signs,* aside from the ascites, are not always satisfactory, though the glandular enlargements may occasionally be detected by palpation. In case of adhesions the exudate may be localized and thus detected by physical examination but the knotting of intestines is often confusing in this respect. Sacculated exudations are sometimes revealed in the pelvic or umbilical regions. Omental tumors, created by shrinkage and curling of the great omentum, are sometimes detected.

Diagnosis.—Much depends upon the discovery of tuberculosis in other organs as the early symptoms are not always suggestive. The abdominal pain, tenderness, ascites, anemia and emaciation are guiding symptoms and if the typical fever is present, this will be of diagnostic assistance. The rectal mucus and the urine may reveal the presence of bacilli. The acute form may be mistaken for *internal hernia,* though the sudden appearance of a localized pain, stercoraceous vomiting, constipation and tympanites, with the absence of ascites, should help to differentiate. *Enteritis* will be differentiated chiefly by the copious mucus evacuations and the absence of ascites. Tuberculosis in other organs is not discovered and no evidence of an abdominal lesion of this kind can be found. *Cancerous peritonitis* may simulate this closely, but the cancerous cachexia soon excludes the idea of tuberculosis; the absence of other tuberculous lesions and the gradually developing tumor will settle the doubt. It prevails also in older subjects.

Tuberculosis of the Liver.—The liver is affected in the majority of cases of miliary tuberculosis, though the granulations are very small and frequently overlooked. It is equally common as a secondary involvement following tuberculosis of the lungs, peritoneum or lymphatics. When these appear as disseminated lesions the liver is enlarged, pale and fatty, and the structure on section is hard and resisting. When caseation takes place small or large cavities may be found underneath the capsule as the result of the degeneration of these caseous tubercles. These also have a tendency to appear in the finer bile vessels. Aside from the enlargement of the liver irregular prominences are detected by palpation and evidences of perihepatitis are usually found. Portal disturbances may appear, but ascites is a rare symptom.

Tuberculosis of the Genito-Urinary System.—*Tuberculosis of the kidney* may occur as a feature of a general miliary tuberculosis or it may be secondary to lesions which start in the prostate, the bladder, ureters or pelvis of the kidney. In the latter instance the disease becomes localized in the renal structure and undergoes caseation until the organ is transformed into a cheesy mass.

The *symptoms* of the miliary involvement are not marked or they may simulate those of nephro-lithiasis. In the primary form there are often no distinctive signs of the disease, sensations of fullness and tenderness in the renal region being the only symptoms. Frequent micturition and pain in the bladder region are more common symptoms and the urine may be purulent and bloody. Cheesy masses with tubercle bacilli are diagnostic findings and with these we shall usually find shreds of fibrous and elastic tissue which evidence a renal-tissue disintegration. None of these symptoms is constant and at times the urine may be decidedly clear. The signs of tuberculous development in neighboring parts like the prostate gland, bladder, ureters, the ovaries and tubes in the female are substantiating symptoms. Secondary pulmonary involvement is to be expected and hydronephrosis may rsult from the obstruction of the ureters.

Tuberculosis of the pelvis of kidney, ureters and bladder is not readily distinguished from a similar involvement of the renal substance and is nearly always secondary to it. The bacteriological examination for bacilli will be most conclusive when obtained, but the symptomatology may point only to those features which are indicative of an inflammatory invasion of the renal tract. The presence of the bacillus unfortunately does not indicate whether it comes from the urinary tract or from the kidney. Cystoscopic examination may be necessary for this determination and even that is not always applicable. Cystitis is generally a common symptom and the urine is invariably purulent and often contains blood. These symptoms may appear when the kidney alone is involved, but the cystitis is not so pronounced. A simple form of inflammation may, however, give rise to similar suggestions; for that reason it will be necessary to exclude the presence of calculus, gonorrheal infection, and also inflammations of the generative tract of the female. The presence of tuberculosis in neighboring parts or in the lungs will aid the diagnosis.

Tuberculosis of the ovaries, Fallopian tubes and *uterus* is not infrequent. The ovaries may contain miliary tubercles or reveal a cheesy degeneration from this cause, though the lesion is usually secondary to a tuberculosis of the tubes. The symptoms simulate those of simple ovaritis. When the tubes are the seat of tuberculous invasion the infiltration may be discovered by the usual method of palpation, while the presence of pus and cheesy masses will be detected by operation, both tubes usually being affected. The uterus, when affected by miliary tubercles, will give evidence of metritis and when softening and ulceration take place it will be possible to find the bacilli in the discharges. The disease may start in the tubes near the fundus or it may extend upward from an involvement of the vagina and cervix. The symptoms of metritis, enlargement of the uterus, together with the febrile signs of tuberculosis, must be looked for. Peritoneal involvement will be a primary or secondary complication.

Tuberculosis of the testes, prostate gland and seminal vesicles.—The testes are implicated secondarily to prostatic and bladder tuberculosis and the glands are enlarged as the process of ulceration and cheesy degeneration takes place. Unless there is active inflammation, with much enlargement, pain is not a marked symptom. The prostate and the seminal vesicles are generally involved together and the symptoms are similar to those which prevail in any inflammatory enlargement of the prostate.

Tuberculosis of the Mammary Glands.—This is of rare occurrence, the bacilli being transported by the blood to these parts from adjacent organs. It is most frequently observed in women during the child-bearing period or in later life. Pyogenic infection may attend the cheesy transformation and when excessive softening takes place fistulae may exist. Sharp pain attends the morbid development and the tuberculous nodules may be distinguished by palpation, while enlargement of the axillary glands is usually a secondary development. The discovery of bacilli in the broken down tissue, together with evidence of tuberculosis elsewhere, will be confirmatory.

Tuberculosis of the Brain.—This has been observed as an acute meningitis of tuberculous character already described elsewhere. It also takes place as a chronic localized infection involving the meninges and the cortex, giving rise to meningo-encephalitis. It generally occurs as a single nodule which develops into a small or irregular mass attached to the brain structure or separated from the same by cysts. The outer zone presents a connective-tissue increase while the central part is caseous and later may become purulent and form a small cavity. The inferior portions of the brain are most frequently affected. Cerebral softening may follow as the result of thrombosis from compression and a tuberculous meningitis may be a sequence. Tuberculosis elsewhere is frequently discovered. The young are mostly affected and the symptoms resemble those of brain tumor. Tuberculosis of *the spinal cord,* with the lesion of a solitary tubercle, is rare, and is secondary in a majority of cases. Acute tuberculous meningitis of the cerebro-spinal form is mentioned under diseases of the cord.

Tuberculosis of the Heart and Blood Vessels.—The *myocardium* is occasionally invaded by the bacilli transmitted via the lymphatics from another focus or more rarely by the blood or directly from the pericardium. It may occur in the miliary form, as a diffuse infiltration or as large tubercles. "The smaller nodules are found usually in the ventricles and the larger masses in the auricles and chiefly the right." No symptoms are positively suggestive of the disease and, therefore, a diagnosis is not easily made. The presence of tuberculosis in other parts may possibly cause suspicion when cardiac failure appears. Death may suddenly follow an attack of syncope. The *endocardium* is affected even less frequently than the myocardium. It may follow a metastasis from the tuberculous enlargement of the mediastinal glands or the infection may rarely occur through the circulation. Vegetations associated with pyogenic infection are usually found on the valves and, in very rare instances, caseous tubercles are discovered. The *blood vessels* are invaded by the tuberculous process through the extension from adjacent parts and through the circulation. The walls are weakened sufficiently to allow perforation and hemorrhage.

Diagnosis of Tuberculosis.—Sufficient is known regarding the pathology and the characteristics of this disease to render the macroscopic and microscopic findings invaluable in making the diagnosis. If we add to these the discovery of the bacillus, all doubt is absolutely removed. The physical examination—particularly in pulmonary tuberculosis—together with the careful differentiations, which must be made in every diagnosis, will furnish evidence which should not be neglected. A true study of the symptoms and a careful watching of every suspected case from the time of its incipiency will furnish additional help. A due regard for the family history may help the decision or at least cause a reasonable suspicion of tuberculosis in many cases. The tuberculin test is probably our most satisfactory adjuvant of diagnosis and this should be employed in suspected cases.

To be of value it must be conducted properly. The temperature of the patient should be taken at two hour intervals and recorded. Then one milligram of a reliable preparation of Koch's old tuberculin should be injected hypodermatically in the interscapular region. The temperature of the patient should be taken again every two hours. A positive test will be indicated by a reaction consisting in a rise of temperature of at least two degrees, but not above 102.5° F. The rise begins in about six hours, reaches its height typically in eighteen hours and continues about thirty-six hours in all. The pulse and respiration rise with the temperature and are accompanied by any two of the following symptoms: Chilliness, headache, nausea or muscle pains. A negative test shows no reaction and it is advised that the rectal temperature is least liable to error. If negative, the test should be repeated after intervals of one or two days with two, three, four and five milligrams of tuberculin successively. Always begin with the smallest dose. If no reaction occurs after five milligrams, the result may be considered conclusive that tuberculosis is not present.

The test is no more dangerous than the use of chloroform or ether for diagnostic purposes, and ranks in value with the Widal typhoid test.

Tetan's method: Take the mean of the morning and evening temperature for twelve successive days. If over 99.5° F., it means tuberculosis or a strong predisposition to it.

Prognosis.—The prognosis in this disease should always be carefully weighed, much dependence being placed on the stage of its activity, the general physical resistance and the heredity. The severity of the disease must be judged by its progress and duration. The extent of the tuberculous lesion is also a deciding factor, for a large cavity in the lungs can only be regarded as very unfavorable. We must, however, keep in mind the fact that a spontaneous arrest of the disease is a possibility. The tubercle bacilli, if left to their natural tendency and not encouraged by the proper soil or other favorable conditions, may be self-limited. The mixed infection is to be most feared but, in the absence of this, with favorable environment and persistent outdoor treatment, we have an opportunity which has not heretofore been appreciated in combating this dread disease. The danger signs should be known by all physicians and a due appreciation of these will make our prognosis more perfect.

Associate diseases and physical debility always predispose to an unfavorable result. Nephritis, cardiac diseases, and gastro-intestinal disorders always diminish the chances for recovery. All of the infections are malicious agents in this respect and nothing renders the diagnosis more unfavorable in pulmonary tuberculosis than the sudden appearance of pneumonia. A previous attack of pneumonia is a natural predisposition to the pulmonary form of tuberculosis. The greatest number of deaths is bound to occur among the poor whose surroundings are unfavorable and the danger of a general spread of the disease from such quarters is always to be feared. Age is always a matter of consideration in making a diagnosis, inasmuch as the mortality, both for the acute and chronic form, is greater in the younger subjects. Under all circumstances great caution should be exercised against making a positive prognosis in any case. So much depends upon the general physical condition, which we do not always understand, and so many mitigating conditions may arise that we must always be on guard in this respect. The disease is at best a treacherous one and a fatal result is to be expected unless the persistent outdoor treatment is instituted under the most favorable conditions.

The Treatment of Tuberculosis.—The treatment of this disease has undergone such radical changes that a long chapter on this subject is now no longer needed. The most advanced scientific deductions which have supplemented careful pathologic research has taught us that the best remedy, and in fact the only true remedy, is found in the pure air which Nature has given us. So long as we defile this and shut ourselves up in poorly ventilated rooms we subject ourselves to the ravages of this disease and thwart its cure when once it is contracted. A proper consideration for perfect health which constitutes the real resisting power, and the overcoming of habits and indulgences which limit this and impair our general health are essentials of paramount importance. Diseases which are associate and predisposing to physical infirmity, should receive unusual attention in their incipiency, while exposure to the tuberculous infection should always be guarded against. Our knowledge of the dangers of the disease is thoroughly established, but we are too frequently derelict, as physicians, in utilizing our therapeutic agents in the early stages of the disease. The tendency of Nature to resist the disease by the process of calcification and fibrosis should lead us to help this effort so far as in us lies. When the tubercles become involuted or quiescent, we should exert our therapeutic influence to the end that we may limit the tuberculous invasion. The sphere of a physician's usefulness in the treatment of this disease is, therefore, greater than a passing thought would indicate.

The *prophylaxis* is an important therapeutic principle, inasmuch as our object should be to limit the contagion as well as to treat the pathologic condition. This embraces not alone the care of the sputum by the employment of an expectoration cup, which may be destroyed or sterilized, but it includes as well the constant use of a proper disinfectant for the sputum. *Segregation* should be demanded under all circumstances, as patients should be controlled so as not to endanger others. The furnishings of the room should be regularly disinfected. Autoinfection should be guarded against and to this end he should be warned of the danger in swallowing his sputum. Conveyance of the infection, particularly by the act of kissing, should be rigorously guarded against. The *registration* of tuberculous patients will surely come to pass when we all realize what it means in protection to the general public. A physician who conceals information of this kind from the public authorities should be condemned for his lack of interest in the public welfare. The work now being done by societies and Boards of Health to popularize the knowledge of this infection and to protect mankind as a whole should receive our most ardent support. The inspection of our food supply is most essential and all sanitary regulations, which are necessary for the limitation of this disease, should have the support, not alone of the profession, but of every right-minded citizen as well.

The *predisposition* to the disease, whether inherited or acquired, should receive careful and prompt attention. This implies more thought relative to food, habits, environment and the early treatment of diseases which favorably predispose. It pertains to the early climatic change, the prompt treatment of incipient symptoms and the outdoor life and physical development which are essential to resist the infection. The early cure of colds and pneumonic invasions are not all that is required, but the removal of tuberculous glands and necrotic bone tissue will demand the services of a skilled surgeon. The convalescing period of an infectious or debilitating disease will require the most painstaking treatment lest a tuberculous infection is thereby encouraged. A tuberculous focus is always a source of danger until it is completely removed.

The *treatment* of tuberculosis itself has not been a satisfactory procedure except by climatic changes or the outdoor treatment persistently applied. The older method of using codliver oil to correct an advanced phthisis has been properly discarded except as it may be demanded for its food value. The *destruction of the bacilli* within the body has been the object of the profession for a long time, but, as yet, no satisfactory result has been obtained. *Antiseptic inhalations* in the form of iodin, creosote, carbolic acid, formaldehyde and other preparations have been assiduously employed, but, with the possible exception of relieving local irritations, no pronounced success has been attained by this method. The use of *internal parasiticides* has also been equally unsatisfactory except as they may be useful in modifying the soil conditions or relieving some of the attending symptoms. *Creosote* has been the most popular of these agents and it has been employed, in increasing doses, to the point of gastric toleration. It may be administered in milk, egg-albumen, or better in capsule, while many preparations of this with codliver oil, etc., have been popular. It has a tendency to allay the cough and in some instances it corrects the fever, but sufficient data is not at hand to prove its efficiency, and it may impair the digestion. *Guaiacol,* in my experience, has been far more satisfactory, and I have invariably used it in five to ten-drop doses in milk, after meals. *Tuberculin* was at first mentioned as a probable successful agent, but its use, so far as we know, pertains only to its diagnostic value.

The *climatic treatment* is our best means of cure in the management of this disease in all its forms. The sooner we institute this in the incipient cases the more sure is it to be successful. Sufficient is already known of the value of this method to permit an abbreviated statement in regard to it. The greatest danger is that both physician and patient are inclined to delay in adopting it. The locality which furnishes the greatest purity and dryness of the air will be most efficient and the equability of temperature is always desired. The rarefied air of high altitudes is best of all, but these localities must be chosen with due regard to cardiac conditions and atmospheric pressure. In advanced stages with hemorrhagic tendencies such signs should be sought by gradual tests of lung and heart endurance. The greatest error committed by patients in such changes from the home altitude exists in the fact that a majority of such people have an ill formed idea that the principle of "roughing it" consists in violent and constant exercise. To best obtain the good effect of such change it is essential, in the majority of instances, to secure constant rest. The patients should sit out of doors in a comfortable reclining chair or lie in a hammock, while it is always better to sleep out of doors or with the windows wide open. At such times no stimulants or drugs should be used unless specially indicated, and the habits and diet of the patient should be most conducive to physical restoration and nutritional improvement. Sunshine is always to be sought, as darkness and dampness favor bacterial development of infectious diseases. Such regions as Colorado, Arizona, New Mexico and many of our western States furnish the best climatic qualities. Certain portions of Georgia, California and the Adirondacks are similarly conducive to good results from outdoor camp life, though the individuality of the patient is an important feature to consider. The northern States, particularly Minnesota and the Dakotas, may be sought for the same purpose, though it is often necessary to change to a warmer climate in the winter. This is true of some of our western States, like Colorado, for in such localities the patients are liable to pneumonia in the colder seasons. *Out door tent life* at home should be tried as a first resort when patients are not able to avail themselves of a climatic change and this has been demonstrated conclusively by the tent colonies which have been established in many towns to be of great value. Of course, this is not applicable in damp localities where fogs frequently prevail. It will soon be necessary to more thoroughly institute these home treatments inasmuch as the western States are already beginning to restrict the emigration of tuberculous patients to their localities. The *sanatorium* treatment, which has been successfully instituted in different States by public and private enterprise, has obtained the most encouraging results. In these special hospitals it is possible to carry out sytematic treatment, hygienic, dietetic and climatic, which can not be accomplished elsewhere.

The *dietetic treatment* is of utmost importance as the nutrition must be maintained or enhanced at all hazards. To accomplish this all remedies which disturb digestion should be proscribed. Forced feeding may be instituted if the patient can digest and assimilate the food. To accomplish the best result it may be well to administer tonics which will stimulate a better appetite and at the same time give remedies which will aid the digestion. The climate, however, is generally the best stimulant of an appetite. Albuminous foods like "milk, meat, fish and fowl, together with an abundance of fats," may be regarded with favor. The hydrocarbons should be used with care lest the digestion is deranged. Eggs usually give the most nourishment and they constitute the best form of diet for forced feeding. Alcohol should only be used sparingly, both as a food and as a stimulant.

Special remedies may be countenanced under various conditions. There are cases, no doubt, which respond favorably to the use of codliver oil which often sustains the nutrition. The *tuberculin serum* may yet be of value in furnishing immunity against infection, but we have not yet perfected that method of specific treatment. The *hypophosphites* are, no doubt, serviceable in conditions of malnutrition with nervous exhaustion, and the careful employment of *iron* and *arsenic* may serve to combat the extreme anemia. *Iodoform inunctions* are highly recommended by experienced practicians, as are many other remedial agents. In the *acute forms* various remedies will be required according to their symptomatic indications. It is claimed that the advent of such acute diseases as smallpox and rheumatism tend to ameliorate or lessen the effect of tuberculosis. The *cough* is frequently so disturbing that palliation will be temporarily demanded. Also the inhalations of creosote, benzoin, carbolic acid, formalin, alcohol, etc., may be helpful. In like manner the cough mixtures which contain balsam, creosote, iodin and tar may be tolerated. One of the best adjuvants of this kind in my experience being the elixir of heroin and terpin hydrate which may be used in teaspoonful doses every two or three hours until relief is obtained. Expectorants are useful when the expectoration is suppressed, but for this purpose we shall obtain best results from the indicated homeopathic remedies. The *fever* also may require special attention, but this will disappear when the toxemic cause is successfully removed. Internal antipyretics should always be avoided for the cardiac exhaustion which is thereby entailed will overbalance any temporary relief which may be attained by such measures. *Creosote,* to a certain extent, tends to limit the fever, but here again the best results are accomplished by the employment of the indicated homeopathic remedy. Free elimination by lungs, skin, kidneys and bowels should be constantly maintained. *Night sweats,* if not relieved by the sponging, may be helped by the internal administration of atropin (gr. 1/120-1/50) two or possibly three times daily, or in combination with agaricin (gr. 1/8); muscarin (m. 3-5) in one per cent solution, has been recommended for the same purpose. The *gastro-intestinal* disturbances will often require a change in the diet or the suspension of a specific remedy. The homeopathic remedies are of best service here. *Diarrhea* is frequently a most disturbing symptom and this may require dietetic limitation, the use of bismuth, chalk mixture, lead acetate, opium or the high hot enema. Hemoptysis does not usually demand special treatment as the hemorrhage tends to be self-limited. The use of ice internally and locally upon the chest, or, in most extreme cases, the administration of ergot, are all that is required.

The *homeopathic remedies* might seem to be useless when we place so much stress upon the climatic treatment. This is by no means to be implied. The principle of our treatment is supposed to pertain to the symptomatic expression of a pathologic condition and, so far as symptoms suggest a condition for treatment, they should guide us in the selection of a remedy. While these remedies may not be regarded as curative agents or in any way antagonistic to the use of a specific agent, their employment will give splendid results. When we forget the symptomatic significance, which means individualized treatment, we indulge in dangerous empiricism. If we hold to this principle and add to it all that science and experience has taught us, we shall better care for our patients while they seek Nature's method of cure. The use of such constitutional remedies as *arsenicum iodid, calcarea phos., calcarea iodid, ferrum phos. and iodoform* will substantiate all the claims that are made for them as associate remedies when they are truly indicated. The *fever* of tuberculosis can not be better managed, aside from specific means, than by the use of such remedies as *aconite, belladonna, bryonia, baptisia* and *echinacea.* The cough will be managed by a host of our indicated remedies, many of which may be enumerated as follows: *Phosphorus, bryonia, hepar sulphur, tartar emetic, silphium, drosera, kali bichromicum, creosote, stannum, sanguinaria, ipecac, spongia, sticta, rumex, iodin* and its compounds, especially the *iodid of antimony, arsenic* and *tin. Hemoptysis* and more excessive hemorrhages, may frequently be overcome by *aconite, ipecac, hamamelis, geranium, secale* and *hydrastin hydrochlorate.* The night sweats will often respond quickly to the homeopathic application of *agaricin, atropin, china, sulphur, eupatorium, phosphoric acid, picrotoxin* and *zinc oxid.* The alimentary disturbances will be corrected by the remedies usually indicated in such conditions and in fact there is no stage or condition of this disease in which we shall not be able to get remedial help from our remedies and our principle of prescribing them. As we care for symptoms we care for disease and in using this method we treat the patient irrespective of the name of the disease, but with due consideration to the diagnosis and the pathology.

SYPHILIS.

This is a chronic infectious disease due to inoculation with a specific virus in acquired syphilis, and to hereditary transmission in the congenital form. The primary lesion, the chancre, appears at the point of inoculation, while the secondary manifestations are recognized by the skin eruption, mucous membrane involvement and glandular enlargement, together with constitutional symptoms. Tertiary lesions affecting the different tissues of the body may appear later, while degenerative changes of the nervous system are a still later sequence. It was formerly thought to be confined to the human species, but recent experiments show that monkeys can be inoculated.

Etiology.—The disease is without doubt the result of *infection* by some micro-organism, though definite knowledge as to its character is not at hand. Lustgarten, in 1884, described the bacillus as resembling that of tuberculosis and leprosy, being "slightly clubbed at the ends and from three to four microns in length." It is a highly contagious disease, the blood of the syphilitic patient being capable of conveying it while the secretions of the primary and secondary lesions are similarly infectious. There is no exemption to susceptibility to the disease and re-infection is a possibility. The physiological secretions of the body do not communicate the disease, unless they are contaminated with syphilitic secretions. "Exceptions to this law are the spermatozoid of man and the ovule of woman, each of which, if derived from the syphilitic source, is capable of infecting the other." (Tyson.) *Predisposing conditions* must favor the inoculation inasmuch as an abrasion of the cutaneous or mucous surfaces, particularly on the genital or oral mucosae, are vital in the contraction of the disease.

The *mode* of *infection* is usually by sexual contact, though *accidental inoculation* occasionally takes place in surgical or medical practice where the surgeon or his instruments convey the infection. The act of kissing by a syphilitic may infect the oral mucosa while eating utensils and drinking cups, together with other articles which are daily handled, may be the media of inoculating exposed mucous membranes or skin abrasions. Certain sexual perversions may lead to infection of the mouth and tonsils. *Hereditary transmission* may take place by paternal inoculation of the fetus through the spermatozoid or ovum, or, more rarely, a syphilitic placenta.

Pathology.—The *primary lesion,* or chancre, appears at the site of inoculation. The structural change is represented by an infiltration of the connective tissue with round cells and a layer of epithelioid and giant-cells. The latter undergoes ulceration, and has a characteristic hard base due to sclerosis of small blood vessels and to cellular infiltration. The *secondary lesions* are characterized by skin eruptions and mucous patches, the latter being usually located at the junction of muco-cutaneous surfaces. The connective-tissue infiltration and the sclerosis of blood vessels are similar to those of the primary lesion. Lymphatic enlargement, falling of the hair, iritis and other symptoms are typical of this stage. The *tertiary lesions* are characterized by circumscribed tumors or diffuse infiltration known as gummata, which are found in all the tissues of the body, not excepting the osseous structure; they vary in size from microscopic tumors to those the size of a hen's egg; they are usually firm and hard, but may undergo rapid disintegration and ulcerate. On section the gummata have a grayish-white color and a homogeneous structure, though the center may present a caseous degeneration while the periphery is fibrous. In some tissues the ulceration and sloughing may be extreme.

Symptoms of Acquired Syphilis.—In the *primary stage* the initial lesion or chancre develops two to four weeks after exposure. It starts as a red papule which slowly enlarges, undergoes central necrosis and finally ulcerates. The surrounding tissue becomes very hard which accounts for the term "hard chancre." A small chancre is often overlooked, particularly when located just within the urethra. The neighboring glands may show signs of enlargement but other symptoms are not present.

The *secondary stage* is recognized anywhere from four weeks to three months after the initial lesion. It is first characterized by a moderate fever (101°-103° F.) with malaise, myalgia and other constitutional symptoms. *Lymphatic enlargement* soon becomes a marked feature, the post-cervical glands being affected early. The *skin eruption* is first recognized as a macular syphilide or a syphilitic roseola, appearing most commonly on the trunk, buttocks, thighs and forehead. These are reddish brown in color and symmetrically arranged. They usually remain for two or three weeks, though relapses may occur after their disappearance. The *papular syphilide* may appear on the face and trunk while a pustular rash which closely simulates variola is occasionally seen later, and a *squamous syphilide* similar to psoriasis, but having more of a copper color, is not rare. *Mucous patches* are visible on the inner surface of the cheeks, on the tongue, lips, tonsils, hard and soft palate and pharynx, as well as upon the genital organs. Further marked features are the symmetry of their arrangement, their rounded outline, their copper color, their lack of pain and their polymorphous character. *Other lesions* are recognized as alopecia, either in patches or as a gradual thinning process, syphilitic onychia, the loss of eyelashes and eyebrows, laryngitis, iritis, choroiditis, retinitis and rarely epididymitis and parotitis. Syphilitic warts or condylomata, due to the hypertrophy of papillae, are often found at the muco-cutaneous outlets especially about the genital organs and anus. Anemia is occasionally a symptom of this stage, giving rise to the so-called syphilitic cachexia. The symptoms of the secondary stage continue from one to three months according to the extent of the lesion, the condition of the system and the promptness of thorough antisyphilitic treatment. Succeeding this there may be, for a time, no apparent evidence of the disease though the symptoms of the next stage may be intermingled.

The *tertiary stage,* while often wanting in truly distinctive symptoms, is usually characterized by the appearance of syphilides which have a tendency to ulcerate. As healing takes place scars are left and these are distinguished by their irregular arrangement compared to the symmetry of those of the previous stage. One form of the tertiary syphilides is known as *rupia,* which is first pustular, then ulcerative and is covered with laminated crusts. A marked scar follows the healing of these lesions. Another form of eruption in this stage is the *tubercular* variety which appears on the face, back and legs. It involves the true skin and, on healing, leaves a scar but its distribution is not symmetrical like the secondary eruption. This syphilide is not believed to be infectious. *Gummata* also develop in the true skin, sub-cutaneous tissue and internal viscera. These, undergoing necrosis, form irregular kidney-shaped ulcers which do not heal readily. When these are found on the mucous membranes they may heal by forming cicatrices with resulting stenosis of the larynx, trachea or rectum. When the muscles are affected small and hard tumors may be formed as the result of the fibroid change. When the bones are affected "nodes" are formed which are painful to pressure. *Amyloid degeneration* is another form of the syphilitic lesions and this is often associated with rectal syphilis in the female. *Parasyphilitic lesions* are those which are not formed exclusively by syphilis and, therefore, do not respond to the anti-syphilitic treatment. They are chiefly recognized as tabes dorsalis, paralytica dementia, epilepsy and arteriosclerosis. A *malignant syphilis* is one of virulent type with no marked stages. It runs a rapid course and responds to no treatment.

Symptoms of Congenital Syphilis.—This form of syphilis may present all the symptoms of the acquired form minus the primary chancre. The signs are apparent at birth, or more frequently during the first four months after birth. In the *new born* the symptoms are evidenced by the impaired physical development, the emaciation, and the skin eruption in the form of bullae on the palms and soles, which are characterized as "pemphigus neonatorum." The child is troubled with "snuffles," while careful inspection will usually reveal fissures and ulcers at the muco-cutaneous outlets. The spleen and liver are enlarged and gummata may be found on the bones while the long bones may show a syphilitic epiphysitis. A pseudo-paralysis occasionally develops.

Early postnatal symptoms appear any time between one and five months. A nasal catarrh, denoting a syphilitic rhinitis and giving rise to "snuffles," is usually the first suggestive symptom. This is usually a sero-purulent or bloody discharge which interferes with respiration and makes nursing difficult. Syphilitic ulceration may cause necrosis of the nasal septum, giving rise to the characteristic "saddle nose." Otitis media and otorrhea may be a development secondary to the coryza and result in deafness.

The *cutaneous symptoms* appear soon after the discovery of the snuffles, usually being defined as reddish-brown patches with well defined outline. The nates are usually first affected and the eruption may resemble a simple erythema or an eczema. A papular syphilide in this region is occasionally found. Fissures about the lips, generally at the angles of the mouth, are characteristic signs. The muco-cutaneous surfaces are the seat of ulcerations and the secretion from these is virulent and infectious. The hair, if the child has any, may fall out, as may the eyebrows. Syphilitic onychia may be detected and there is moderate enlargement of the glands. Splenic and hepatic enlargement is sometimes detected, though these changes may be associated with other conditions. Hemorrhages of a mucous or subcutaneous character, or from the umbilicus at birth, may occur, though these are not alone diagnostic of syphilis. The child is usually anemic, restless, irritable and inclined to cry out frequently during sleep.

Later manifestations of congenital syphilis will be indicated to a decided extent by the general appearance. Such children rarely thrive and they present a wasted and warped appearance which is suggestive of premature old age. The skin has an earthy tint, the hair does not grow, the nourishment seems deficient, the functions are impaired and after a few years the child looks much older than he is. The skull is asymmetrical, the forehead being prominent with the frontal eminences marked. The lips are prominent, the bridge of the nose is often depressed, the tibia may be "saber-shaped" on account of a gummatous periostitis, and a chicken-breasted thorax may be observed. The teeth do not develop properly, presenting various irregularities and the dental arch is not perfectly formed. "The incisors of the permanent set are notched, the free edges are thin, the summit is atrophied with crescent shaped erosions." This dental distrophy is considered by many as a marked diagnostic sign of inherited syphilis. Iritis and interstitial keratitis are occasionally detected while deafness is a common sequence of the otorrhea. The testicles are usually undeveloped. Multiple round or serpiginous cutaneous scars are often found over a circumscribed area. Symmetrical synovitis may rarely affect the knees. Enlargement of the spleen and induration of the lymph glands may occasionally be associated with hepatic enlargement.

Visceral Syphilis.—*Brain* and *cord* lesion may be found as follows: (1) Gummata in the form of multiple or single tumors are attached to the pia, and often the dura, extending secondarily into the brain tissue. In size they vary from that of a millet-seed to the dimensions of a hen's egg. When small they have a "uniform and translucent appearance, but when large the center undergoes a fibro-caseous change, while at the periphery there is a firm, translucent, grayish tissue, being more commonly found in the cerebrum." They may resemble tuberculous growths the differentiation being made by their uneven surfaces and the more irregular caseation. When the membranes are involved, a subacute or chronic meningitis may follow. A diffuse meningitis often extends from the pia to the dura and the brain substance. Cystic degeneration of the gummata is occasionally observed. Gummatous growths may involve the cord, but these, in the large form, are not so commonly observed. (2) *Endarteritis* is a lesion which results in aneurism, hemorrhage or a narrowing and obliteration of the lumen of the blood vessels. Cerebral thrombosis, softening and secondary degenerations are the common results of this invasion both in the cord and the brain.

The *etiology* points to the fact that the majority of these lesions are the result of acquired syphilis. In rare instances cerebral gummata may develop from the inherited form between birth and puberty. The most frequent period of occurrence is between the third and fifth years, the lesions usually developing within twenty-five or thirty years after infection. In many instances the evidence of primary or secondary symptoms may have been overlooked.

The *symptoms* of the inherited form are often defined only by imbecility and idiocy, plus many of the features of the acquired form. In the acquired form, aside from the psychical features characterized by delirium, headache and convulsions which may be recognized in the early stages, the symptomatology is referable to a condition of epilepsy, brain tumor and paralysis. *Epilepsy* appearing late in life, or at least after the twenty-fifth year, may be presumably of syphilitic origin, other signs of which should be carefully looked for. In so many cases such history is denied and it is difficult to make a decision. The presence of scars and bone lesions will help in distinguishing this from other forms of epilepsy. The accentuated psychic disturbances and the hysterical manifestations together with a protracted torpor may be suggestive. It will always be necessary to carefully differentiate the symptoms of idiopathic epilepsy.

A *brain tumor* of syphilitic origin is not easily determined unless we find the lesions of the specific disease or obtain a history which substantiates such a diagnosis. The symptoms are the same as those given under this subject in the diseases of the nervous system. If a primary chancre is admitted and evidence of scars and specific degeneration is obtained we may reasonably suspect a syphilitic growth when the characteristic symptoms of brain tumor, headache, optic neuritis, convulsions and paralysis, appear. Some confirmation of this diagnosis is elicited by the improvement which may result from antisyphilitic treatment. The *paralysis* which follows cerebral hemorrhage or the presence of a gumma may appear as hemiplegia or paralytic dementia. *Cord in-*

volvement by a gumma does not give a very clear group of symptoms. Myelitis, acute or chronic, and most frequently the latter, is the most characteristic development. This appears generally within five years after the initial lesion, the lumbodorsal cord being most frequently affected. The symptoms other than the scars which are confirmed by the specific history are not distinctive while the antisyphilitic treatment gives but little result. The prognosis in both syphilis of the brain and cord are exceedingly unfavorable, more particularly so in acute myelitis. The *general diagnosis* in syphilitic invasion of nerve tissue will rest upon signs of focal lesions plus the multiform and anomalous symptoms which pertain both to the brain and the cord. The history and the improvement attained by antisyphilitic treatment will be specially confirmatory.

Syphilis of the liver is observed in three different forms. (1) A *diffuse syphilitic hepatitis* is commonly a congenital affection. The liver may retain its normal shape but is uniformly enlarged and hard. The color is grayish-yellow or sometimes it assumes an amyloid appearance. Microscopic examination reveals a decided increase of connective-tissue with foci of small-celled infiltration. In case there is extensive cicatrization there may be some deformity and the cicatricial increase is liable to reduce the size of the liver. (2) *Gummata* usually develop as tertiary lesions two to six years after the initial lesion. They appear as disseminated nodules which have a central cheesy mass with a peripheral fibrous zone. The contractions which follow these tend to reduce the size of the liver while deep contracting fibres may be seen throughout the hepatic structure. Aside from the general fibrous change the gumma may soften and liquify. Previous to the contraction there may be an increase in the size of the organ with occasional protruding points. In congenital syphilis miliary gummata are found. (3) A *gummatous arteritis* may be found in the portal vein and the hepatic artery though the smaller arteries more commonly are involved. (4) A *perihepatitis* may exist as the result of a thickening of Glisson's capsule following a specific infection. The connective-tissue increase may invade the deeper hepatic tissue by the extension of cicatricial bands.

The *symptoms* are not always indicative of the true condition. In the *congenital form* hepatic enlargement is in evidence and the same is generally true of the spleen. Icterus is usually recognized. With this we may find some signs of cutaneous or bone lesions while evidence of specific infection may be found elsewhere. In the adult, syphilis of the liver will generally be detected by the signs which simulate hepatic cirrhosis. Icterus is slight. The contraction naturally induces ascites together with splenic enlargement the same as in hepatic cirrhosis. Gastro-intestinal disturbances, pain in the right hypochondrium, some tenderness on pressure and a general loss of weight will also be characteristic symptoms. The enlargement and the irregular protuberances may often be detected by palpation in the early stages before ascites has developed, or after withdrawal of the fluid. *Amyloid changes* may take place in the liver and simultaneously in the spleen. Enlargement of both of these organs may be discovered on a careful examination and the anemia due to such degeneration may be manifest. The urine is usually increased in quantity, albumin and tube casts being found.

A correct *diagnosis* in syphilis of the liver must be made for the sake of the treatment. Much dependence must be placed upon the history and the discovery of scars in the throat or upon the skin and the irregular nodosities on the tibia. The irregular enlargement of the liver is a guiding sign and the general good health is an exception in the majority of hepatic diseases. *Echinococcus cysts* may be suggestive of this disease, but the closest resemblance is found in cancer of the liver and cirrhosis. *Cancer* is, however, never congenital, it occurs in middle or later life and often furnishes the history of a primary growth elsewhere. In cancer ascites is extremely rare, while jaundice is only an occasional symptom. On palpation cancer of the liver may reveal the presence of nodular and umbilicated nodules which must be compared to the irregular margin of the syphilitic liver. The cachexia which attends hepatic cancer and the gradual decline of health which precedes the fatal termination is not found in syphilis of the liver. In the latter also the course of development is slow and decided improvement, if not cure, may be obtained by the antisyphilitic treatment.

Syphilis of the alimentary canal must also be considered. The signs of syphilis which are found in the mouth have already been referred to. The *tongue* may be the seat of gummata and associated with the disease we may frequently observe a fissured condition and evidences of scars. The *palate* is frequently involved while ulcerative gummata are often located on the posterior wall of the *pharynx,* fatal hemorrhages sometimes arising as the result of erosions. The *esophagus* may be stenosed as the result of syphilitic invasions while syphilitic infiltrations are rarely found in the stomach walls. The *rectum* is the most frequent seat of syphilis so far as the alimentary canal is concerned. The gummata develop in the submucosa above the internal sphincter. Stenosis is gradually developed, the bowels are moved with difficulty and hemorrhages with mucus and pus often attend the evacuations. The narrowing takes place from below upward giving rise to dilatation of the rectum above this point. The disease is distressing not alone from the hemorrhoids and prolapse of the rectal mucosa, but because of the loss of strength and the progressive asthenia which attends.

Syphilis of the lungs is a rare form of visceral syphilis. It may exist as gummatous nodules which are distributed throughout the lung though they are more numerous near the root. They have a caseous-appearing center with the external fibrous zone, while cicatricial bands are interspersed between the nodules and attached to the pleurae as well. Secondary changes may lead to softening, while ulceration and cavity formation, or cicatrization, may lead to recovery. The fibrous infiltration may also assume the character of *interstitial pneumonia,* the lesion being located in one lobe of the lung. Bronchiectasis is frequently a complication. *Fetal pneumonia,* or the white pneumonia of the fetus, is a hepatization of a large area or an entire lung as the result of miliary gummata. The tissue has a grayish-white appearance, the alveolar walls are infiltrated and the air cells are filled with desquamated epithelium.

The *symptoms* in lung syphilis may resemble an ulcerative phthisis with the absence of bacilli, one case of which I have seen in my practice. On the other hand the symptoms may point to fibroid induration with a syphilitic history. Otherwise the clinical signs are not prominent. The *diagnosis* will rest upon the history and the improvement obtained by antisyphilitic treatment. Pulmonary tuberculosis will be differentiated only by microscopic examination, and bronchiectasis may characterize the symptoms when a peribronchitis follows a syphilitic lesion of the lung.

Syphilis of the Circulatory System.—The *heart* may suffer from a fibro-sclerotic myocarditis as the result of a gummatous invasion of the wall of the left ventricle. The *coronaries* suffer from a narrowing of their lumen and their branches are similarly affected. *Syphilitic endocarditis* of the fibro-sclerotic type is also of rare occurrence. The *arteries* may also be affected by an obliterating endarteritis as a result of syphilis which creates a proliferation of sub-endothelial tissue. Gummatous periarteritis is also occasionally observed when the vessels are invaded by nodular masses which start in the adventitia and develop outward. Atheroma and aneurysm are frequently the result of a specific invasion of arterial tissues.

Syphilis of the Kidneys.—Gummata are occasionally found in the kidney following a gummatous hepatitis. The lesions are not generally numerous and cicatrization is the usual result. Acute syphilitic nephritis has been claimed as a lesion of the secondary stage appearing in from three to six months after the initial lesion. Chronic interstitial nephritis and amyloid degeneration have also been observed, the symptoms of the latter being determined by the urinary examination.

Syphilitic Orchitis.—Syphilis affects the testes in two ways: A gummatous mass or multiple nodules may invade the substance of these organs, giving rise to an indurated mass which is painless and does not break down. The other form, known as interstitial orchitis, causes induration with consequent atrophy, involving usually one testicle more than the other.

Syphilis of the Joints.—These have been described by Hutchinson as follows: (1) A synovitis which appears during the secondary stage but soon disappears under the appropriate treatment. (2) Perisynovial gummata which pursues a chronic course. (3) Arthritis due to osseous nodes or gummata in the neighborhood of the joints which is characterized by severe nocturnal pains. (4) A chronic form of synovitis which is of most common occurrence. (5) A syphilitic chondro-arthritis.

Diagnosis of Syphilis.—The diagnosis is not absolutely made in every case as the patient may deny the initial lesion which may have been so slight as to avoid detection. This is frequently the case when the chancre is in the urethra or associated with an attack of gonorrhea. Sometimes the secondary lesions are not marked and a careless physician, relying upon the word of his patient, might overlook them. Much care should be taken in obtaining the history, for this is surely a guiding factor. Next to this we must rely upon the symptoms which have been enumerated and these will be substantiating when they are clearly manifested. *Inspection* may reveal the old lesions in the throat and on the skin, while the nodes may be found on the bones. A copper colored cicatrix, if not due to other causes, will be confirmatory. Abortion in women is also suggestive. In the congenital form the occurrence of snuffles, rash, symmetrical nodes on the tibia, and interstitial keratitis will, together with the stunted growth, naturally direct our attention to this disease.

The *therapeutic test,* by means of antisyphilitic remedies, is valuable to a certain extent, though it does not furnish so much evidence when there are visceral lesions. The *Justus blood* test may be of some value. This consists in a marked though transient reduction of hemoglobin following the inunction or hypodermatic use of mercury. *Differential diagnosis* must establish the difference between the primary lesion of the lips and epithelioma, the cutaneous eruption must not be mistaken for papular eczema and other skin eruptions, and syphilitic arthritis must not be confounded with rheumatic or gouty lesions of the joints.

Treatment.—*Prophylaxis* in the treatment of this disease has an important bearing. The illicit sexual relation which is the most frequent cause of exposure is a difficult matter to overcome and will require not alone more rigid municipal control but a higher education regarding the sexual relations in society. Though such suggestions at first seem to be academic it is not a remote possibility to expect the cultivation of a higher sense of morality. Society owes a duty to mankind in this respect and when once the moral obligation is cultivated it is bound to exert a powerful influence. Vice, if not overcome, must at least be segregated and the spread of the disease must be limited to a certain extent. When once the laity learns of the danger to which all are exposed by the service of those employed in restaurants and other occupations in which contamination is likely, it will be possible to ostracize many who expose us. Let us once establish hospitals for the compulsory isolation of those afflicted and results will be attained equal to those established by the isolation hospital in the treatment of smallpox. The marriage of one afflicted with syphilis should be interdicted if possible. *Treatment of the primary sore* has been regarded with varying views. Many still believe in thorough cauterization, while others believe only in local antisepsis. The latter class of practicians believe the hard chancre is only "an expression of general disease" and that it is amenable only to internal medication, which view is borne out by many able observers. A simple dressing of lint, saturated with mercury bichlorid (1-2000), a dusting powder of equal parts of calomel and acetanilid, or possibly a mercurial ointment, may be sufficient for the local sore, which usually heals in a short time.

The *constitutional treatment* has reached a stage of efficiency which may be regarded as truly specific. For this reason we no longer consider this disease as beyond the possibility of a complete cure. *Mercury* and the *iodids* are most efficient in this respect and, "if properly used, will eradicate the second stage and hold the third in abeyance even after it has manifested itself." "Mercury is the remedy par excellence of the second stage and iodid of potassium of the third." Some, however, regard the latter as efficacious from the beginning of the secondary to the end of the tertiary period. Mercury is probably best employed by the method of inunction and this may be performed as follows: After a warm bath has been taken one dram (4 gms.) of mercurial ointment is daily rubbed into the groin or other parts of the body on succeeding days. The friction should be continued until the skin is perfectly dry, using a full half hour for the massage. The part anointed should be thoroughly cleansed on the following day. The mouth should be carefully washed with a solution of chlorate of potash or other antiseptic solution to avoid salivation. Should this occur the treatment should be suspended for a time, but the daily friction should be kept up. If the signs of mercuralization should continue, and no further evidence of the infection be evident after a time, the inunctions may be discontinued. The *internal use of mercury* should now be continued, the protiodid (gr. 1/4) or the biniodid (gr. 1/16) being employed three or four times daily. The homeopathic preparations in the second or third decimal potencies may be substituted, but I doubt if they are so efficient. The employment of mercury by means of *fumigation* has been recommended but it does not seem to be a satisfactory procedure. To accomplish this a patient sits in a chair wrapped in a blanket so that a tent is formed. Under the chair a spirit lamp is burned and over this a tin plate is placed on which calomel is sprinkled. The mercury is volatized by the heat and deposited with the vapor on the skin of the patient. This procedure is continued for about twenty minutes, after which the patient is covered and put to bed without washing or drying. The *direct injection* of *mercury* into the muscles or veins is another method of its application. One-third of a grain (0.0216 gm.) of bichlorid, dissolved in twenty minims of water, is injected once a week or from one to two grains of calomel (0.066 to 0.132 gm.) in twenty minims of glycerin and water is used. The deeper muscles, like the buttocks, should be selected to avoid the possibility of abscesses, which are the greatest danger. A French preparation known as *cypridol,* being a solution of the biniodid in oil has been successfully employed and does not cause pain as does the watery solution. In *hereditary syphilis* one-tenth grain of calomel is employed in the form of inunction or better in the form of homeopathic preparations.

Iodid of potash is the specific remedy for the tertiary and possibly for earlier stages. This is administered in the saturate solution, well diluted with water, after meals. Five to ten drops are given at first and this may be increased gradually to the point of endurance, as much as twenty to sixty grains often being required in obstinate and severe cases. The appearance of an erythematous rash, coryza, salivation and throat constriction and other unfavorable symptoms, should demand a decreased dose or its temporary discontinuance. If mercury is used in conjunction with potassium iodid, it should be in the form of the biniodid, as the others produce mercurial salivation. The diet should be limited and stimulants should be avoided. The daily employment of a hot bath is essential and the Turkish baths may be tolerated in some instances. The baths of Hot Springs in Arkansas and Virginia are very popular in this respect.

When mercury and the iodids have been abused, in cases of inherited syphilis or when there are positive indications for other remedies, these should be employed according to their symptomatic suggestions, either as intercurrent or direct medicinal agents. So far very few remedies, aside from mercury and the iodids, have been mentioned by experienced practicians. *Aurum chlorid* and *nitric acid* have received some attention, but these have more particular reference to the degenerative changes following the specific disorder. The sequelae of syphilis are often amenable to our homeopathic remedies. Such remedies as *stillingia, phytolacca, aurum mur.,* and *aurum met., arsenicum* and *arsenicum iodid* should be studied.

LEPROSY.

Leprosy is a chronic infectious disease which is caused by the bacillus leprae. It is characterized by tuberculous nodules in the skin giving rise to tuberculous leprosy or by a similar infiltration in the nerves causing an anesthetic leprosy. The disease is of early date, being traced back to the ancient history of Egypt and India, and receiving mention in the Books of Moses. It has become almost extinct in Europe, though it is yet endemic in Oriental countries as well as in the Sandwich Islands, West Indies and some parts of the United States and Canada.

Etiology.—The *bacteriology* was established by Hansen, who discovered the bacillus leprae in 1880. It has a close resemblance to the tubercle bacillus. "The bacilli are delicate rods whose length equals 1/3 to 1/2 the width of a red blood disc. They are for the most part found in the interior of cells, rarely outside of them. Some of these cells are of large size and known as lepra cells. In the interior of these cells the bacilli often form clumps. They are exceedingly numerous in leprous tissue. They stain readily in anilin colors but not in vesuvin, differing in this respect from tubercle bacilli, and also in that they liquify coagulated blood serum while tubercle bacilli do not. In the fresh condition the lepra bacilli exhibit active movement." (Tyson). The *predisposing causes* are not marked, inasmuch as all ages are susceptible between the twentieth and fiftieth years. Sex, latitude and even personal contact, which has always been feared, are not claimed as directly responsible. Hereditary transmission is denied, and, except for the fact that the disease seems to prevail in certain localities, we know very little about it. Morrow claims that the disease spreads by sexual intercourse, though the bacilli are supposed to enter the system through the abrasion of the skin. It was the claim of Hutchins that a fish diet predisposed to the disease.

Pathology.—The tubercle of leprosy is a granular mass composed of various sized cells with a connective-tissue matrix. The bacilli, in large numbers, are found within and between the cells. The nodules gradually invade the skin, developing a tuberculous mass which undergoes ulceration and cicatrization. On the face this produces the so-called "facies leontina." The mucous membrane, particularly of the conjunctiva, the cornea and the larynx may be involved. Glands, cartilages, and different viscera may also be affected. When the ulceration is deep seated the fingers and toes may slough off. The nodes are vascular in this respect differing from tuberculosis. In anesthetic leprosy a peripheral neuritis develops, due to the presence of bacilli in the nerve fibres and, as a result of this, perversions of sensation and trophic skin changes are recognized.

Symptoms.—If there is an *incubation* period it is not usually recognized. The *onset* may be characterized by a slight intermittent fever of long duration. There is at first a patchy erythema of the skin which later fades out, leaving white areas (lepra alba). This gives rise to the term "macular leprosy," and frequently the disease makes no progress beyond this stage. The *tuberculous form* is more common and this is characterized by the infiltration of the skin by tubercular nodules. These nodules may retain their form, without degeneration, for a long time, but eventually ulceration and softening take place. The nodules vary in number, size and character, some of them often disappearing. The infiltration is in some parts like the eyelids, nose and ear, merely a thickening, small areas of which may undergo ulceration. When the cornea and conjunctiva are the seat of these nodules blindness may follow. These leprous deposits may be found in the lymphatic glands, the liver, lungs and other internal viscera. They are also found in the mucous membranes, often producing obstructions to the respiratory passages.

In the *anesthetic form* the peripheral nerves become infiltrated with the leprous growths, the nerves becoming hard and nodular. Pain, which is a symptom of this variety, soon gives place to anesthesia, while trophic perversions cause excessive dryness, smoothness and tightness of the skin, the nodules often disappearing. The atrophy which ensues in the fingers and the toes and even the larger limbs may cause a dry gangrene and consequent sloughing. Sometimes large vesicles develop which may be attended by edema of the parts involved. A general weakness and progressive exhaustion gradually develops and the patient dies from asthenia.

Diagnosis.—The diagnosis depends upon the discovery of lepra bacilli in portions of the tissue which have been removed. *Scleroderma* simulates the anesthetic variety but the trophic changes are not so prominent in the former disease. *Syringomyelia* bears a close resemblance to the anesthetic variety. The former disease has no febrile manifestation and its pathology is located in the upper part of the spinal cord. The upper extremities are, therefore, first affected, the lower extremities are involved very little if at all and the face usually escapes. Sweating is also a marked feature of syringomyelia. In this the proximal portions of the extremities are affected, while the distal portions are involved in leprosy. In syringomyelia the anesthesia is manifested upon the trunk in the form of a girdle, the sensory disturbances always bearing a definite relation to the part of the cord involved and the trophic perversions are not so severe as in leprosy.

Prognosis.—While death results only from a gradual exhaustion the disease responds to no known treatment. The patient may die from some intercurrent disease, but in the anesthetic variety a cure is possible though the secondary changes are permanent.

Treatment.—As yet no line of treatment has been carried to a successful end and segregation is the only hope in this disease. *Potassium iodid, creosote* and *mercury* have been advised but no success has been gained from their use. *Salicylic acid, chaulmoogra oil,* in two dram (8 gm.) doses every two hours, and *gurjum oil* have been recommended.

GLANDERS.

This is an infectious disease in the horse which is occasionally communicated to man. It is characterized by nodular growths in the nares (glanders) and beneath the skin (farcy). The disease is due to the bacillus mallei which was described by Loeffler and Schütz in 1882. It is a "short, non-mobile bacillus similar to that of tubercle and leprosy but shorter than either. It is commonly seen among the cells of the growth but has also been found in the blood." Infection is obtained by conveyance of the purulent or catarrhal discharges from the animal's nares to an abraded cutaneous surface or possibly to a mucous membrane. In men the disease is observed only among those who are about horses and it is a decidedly uncommon disease. Transmission from man to man is of rare occurrence.

Pathology.—The new growths have been described by Virchow as granulomata which are characterized by the presence of numerous lymphoid and epithelioid cells in which are found the bacilli. These nodules invade the skin, mucous membranes and muscles. The internal organs may also be affected, the nervous system and cartilages not being exempt. The nodular masses soften and form ulcers in the nasal mucosa, which accounts for the catarrhal discharge, while abscesses develop when they are located subcutaneously.

Symptoms.—Both glanders and farcy are found in the acute and chronic form. *Acute glanders* has a short period of incubation lasting only three or four days. Fever is present and the point of infection shows redness, swelling and lymphangitis. These signs are soon followed by the formation of ulcers with a fetid muco-purulent or blood-streaked discharge and nose-bleed is often observed. An eruption of papules which soon become pustular appears on the face, the trunk and extremities, particularly about the joints. The pustules dry up as fresh ones appear and this is a distinction which compares with varicella rather than variola. The face swells and the nose resembles a bluish-brown tumor covered with vesicles, while the adjacent mucous membranes and occasionally the gastro-intestinal mucosa may become involved. *Chronic glanders* is of rare occurrence with symptoms that are not well defined. Ulcers are often found in the nose, and catarrhal symptoms, plus a bloody muco-purulent discharge, are generally observed. The larynx may also be affected and an irritable cough is occasionally present. Tedeschi has described a case of chronic osteomyelitis followed by a fatal meningitis due to the bacillus mallei. Muscular and arthritic pains, a slight fever at intervals, general asthenia and progressive emaciation are also features of the chronic form of the disease.

Acute farcy follows the inoculation of virus into the skin of man. The symptoms are those of a phlegmonous inflammation of the skin appearing early. The lymphatics are soon affected, nodular subcutaneous enlargements which rapidly suppurate appearing along their course. Abscesses form in the muscles, and the joint pains and swelling cause much suffering. Catarrhal symptoms are not in evidence as the nose is not affected. The constitutional signs closely simulate those of acute pyemia. The disease is generally fatal in a week or ten days. *Chronic farcy* is characterized by granulomatous tumors, which tend to become abscesses without much inflammatory development. The lymphatics are not usually involved but ulcers may form and penetrate the deeper tissues until the bone is sometimes invaded. The abscesses are primarily in the subcutaneous tissue near the joints and these tend to open spontaneously. The disease is inclined to continue its chronic course for several months though death may result from pyemia or the development of acute glanders. The general symptoms resemble those of chronic glanders, the prostration and emaciation usually becoming extreme. The disease may be transmitted from man to man; washerwomen are prone to the infection. The *diagnosis* depends on the history of contact with an affected animal. The injection of "mallein," one of the products of the bacillus mallei, causes a febrile reaction which is diagnostic in degree with the rise in temperature. *Treatment* offers little hope except in the chronic cases which may respond to prompt surgical interference.

ACTINOMYCOSIS.

"Big jaw" and "lumpy jaw" are the other terms which have been applied to this disease. It is more commonly an infectious disease of cattle caused by the actinomycosis or ray-fungus. This develops in the tissues forming new and metastatic growths with a secondary chronic inflammation and pyemic infection. The *parasite* "belongs to the streptothrix group of bacteria and may be seen in the pus of the affected part as yellowish or opaque granules from one-half to two millimeters in diameter. These are made up of cocci and radiating threads, which present bulbous, club-like terminations." The parasites have been cultivated and natural as well as artificial inoculations have been made. The infection is generally transmitted by the flesh and milk of diseased animals while the skin and air passages are supposed to be a means for its entrance in case of an abrasion or an inflammatory condition. The site of infection is usually in the mouth of the neighboring passages. Barley, oats and rye are perhaps a means of its entrance in the case of animals.

Pathology.—The growth of the parasite causes a small granulation tumor which is made up of small round cells, epithelioid elements and giant cells. After a time there is a proliferation of surrounding connective-tissue and the growth, which is often in the jaw, assumes the appearance of an osteosarcoma. Suppuration follows later though in man this may be the first pathological development.

Symptoms.—*Oral actinomycosis* will be characterized by toothache, impaired deglutition, and difficulty in moving the jaw. A swelling soon develops near the angle of the jaw with prompt suppuration and discharge of pus externally and into the mouth. The tongue may be involved but the process usually extends downward, the intestines occasionally being involved. *Pulmonary actinomycosis* is a rare though occasional form of the disease. It is characterized by pain in the side, usually due to pleurisy. Cough attended by a muco-purulent expectoration, fever of irregular type and emaciation are also typical symptoms. The lesions are generally unilateral, though they may be associated with a chronic bronchitis in which case they are bilateral. A miliary actinomycosis or groups of fungi may resemble miliary tubercles. A more extensive destruction of pulmonary tissue may give rise to interstitial changes, abscesses or cavities. Actinomycotic invasion of other organs may exist at the same time. This form of the disease may resemble pulmonary tuberculosis or a fetid bronchitis in its development but a sputum examination will reveal the actinomyces. Rarely it assumes a typhoid character. Death usually comes as the result of septic intoxication. *Intestinal actinomycosis* is caused by the parasitic invasion of the intestinal mucosa resulting in the proliferation of connective-tissue and the formation of submucous nodules. These ulcerate and, should perforation take place, peritonitis follows. The symptoms point to an intestinal catarrh with irregular attacks of diarrhea the parasites being found in the stools. The stomach may partake of the catarrhal disturbance and secondary metastatic growths may cause abscesses in the liver and spleen. *Cutaneous actinomycosis* resembles lupus, the skin presenting chronic suppurating ulcers which contain the ray-fungus.

Diagnosis.—The disease presents the features of a chronic pyema but the presence of the actinomyces determines the diagnosis. Sarcoma of the jaw may resemble this disease but it has a more rapid course and suppuration is not so marked a feature.

Treatment.—Operative removal is absolutely essential in the treatment of this disease. As yet little success has been obtained from internal medicine, though iodid of potash has received some consideration. Billroth cured a case of abdominal actinomycosis with fifteen tuberculin injections.

ANTHRAX.

Anthrax has been described as splenic fever, malignant pustule and wool-sorter's disease. It is an acute infectious disease caused by the bacillus anthracis. It is usually found in animals, particularly sheep and cattle, man being affected by accidental inoculation.

Etiology.—The infectious agent is a non-motile, rod-shaped bacillus, five to twenty-five microns in length and one to one and five-tenths microns in breadth. "The bacilli multiply by fission with great rapidity and are easily grown in various culture media extending into long filaments which interlace and produce a dense net-work. The spore formation is seen with great readiness in these filaments, but an asporogenous variety is known and can be produced artificially in cultures." The bacilli are easily destroyed but the spores are very resistant, withstanding 5 per cent carbolic acid and, for a few minutes, even boiling. The disease is not very common in America, being most frequently observed in European countries. The virus enters the human body through skin abrasions, the intestines and rarely through the lungs. It is claimed that insects may convey the infections.

Symptoms.—Two forms of the disease have been observed, external and internal anthrax. *External anthrax* has two forms. (1) *Malignant pustule,* which is characterized by the appearance at the point of infection of a small papule which develops into a vesicle of considerable size. It is usually seen on exposed surfaces like the hands, arms or face. The vesicle ruptures within thirty-six hours leaving a dark bluish or black scab and this is surrounded by an inflammatory induration with small vesicles. The brawny induration, extending from the base of the eschar, is generally extreme, giving rise to much edema which may extend to adjacent tissues. The neighboring lymphatics are often affected, this being manifest by the red lines, swelling and pain. Fever usually rises rapidly, there is copious perspiration, marked prostration, splenic enlargement and in some cases delirium. In such extreme cases death may take place in a few days. When the constitutional symptoms are milder, the eschar gradually sloughs, the wound heals, and recovery takes place. A fatal collapse is liable to occur in any case in four to six days.

(2) *Malignant anthrax edema* is characterized by an extensive edema without any papule or vesicle following the constitutional symptoms. When the edema is intense gangrene may ensue and grave constitutional symptoms are present. The eyelid, head, hand and arm are the usual areas affected. Endocarditis, enteritis and peritonitis sometimes develop in this form, which is usually fatal.

Internal anthrax is also recognized in two forms, an intestinal mycosis and as wool-sorter's disease. (1) *Intestinal mycosis* is usually announced by a chill, followed by pains in the back and limbs, vomiting, and diarrhea with bloody stools. The infection probably takes place through the stomach and intestines from eating the flesh or drinking the milk of animals affected with this disease. Hemorrhages from the mucous membranes are of occasional occurrence. Dyspnea, cyanosis, extreme nervous symptoms, coma and convulsions generally indicate a fatal termination. (2) *Wool-sorter's disease* occurs among those who sort or handle wool or hair. It is a disease most commonly found in Russia and South America and for that reason is contracted by those who work with imported wool and hair. An external lesion is not observed, the infective agent being swallowed or inhaled. Without premonitory symptoms the patient is seized with a marked chill, pains in the back and limbs with a rapid rise of temperature to 102°

or 103° F. There is great prostration, respiration is labored, and a troublesome cough is indicative of bronchitis. The cardiac action is excited, the pulse is feeble, vomiting is extreme and death may suddenly come without further symptoms. In other instances cerebral symptoms may be present, the patient dying in a state of delirium or coma. Evidence of a co-existing external lesion is sometimes recognized. The duration is from one to five days. *Rag-picker's disease* is another form of the same disease. It is found among the rag-sorters in paper-mills, the infection taking place in the respiratory tract. This seems to be a local anthrax of the lungs and pleurae followed by a general infection which is characterized by the usual symptoms.

Diagnosis.—The character of the lesion in external anthrax is our means of diagnosis though this should be confirmed by a microscopical examination of the pustular contents for the anthrax bacilli. A culture may be made and a guinea-pig or rabbit should be inoculated. Internal anthrax is more difficult but when the patient has been exposed by occupation we may be guided by the pulmonary and gastro-intestinal symptoms plus the signs of systemic intoxication. The presence of the bacilli in the blood should be demonstrated.

Prognosis.—External anthrax may be amenable to surgical measures and in rare instances it may run a favorable course. Internal anthrax is an incurable disease, though in wool-sorters a favorable result may be expected if they survive ten days.

Treatment.—Prophylaxis must consider the destruction of wool, hair and hides which are contaminated and the killing and cremation of the infected animals. Surgical care of the malignant pustules necessitates complete excision, followed by thorough cauterization. Injection of a solution of carbolic acid, water and glycerin (1 to 10) into the surrounding parts has been recommended to limit the advance of the infection. Internally stimulants and antiseptics are indicated, as is free elimination and nourishing food.

HYDROPHOBIA.

Hydrophobia or rabies is an acute infectious disease of animals which is communicated to man by inoculation. It is characterized by an intense tonic spasm beginning in the larynx and pharynx and is attended by slight fever. A fatal termination usually follows coma.

Etiology.—The dog is most frequently the cause of hydrophobia in man, the rabid animal introducing the virus-containing saliva into the open wound made by its bite. The wolf, cat, skunk and other animals are also subject to the disease and may convey it to the human being. The *bacteriology* is not fully understood though there is no doubt of its infectious nature and that the saliva is a medium for its introduction. Pasteur has clearly demonstrated that the poison is resident in the central nervous system, the medulla and brain being its most frequent site.

Pathology.—The morbid anatomy seems to be limited to the upper part of the spinal cord, the medulla, pons and cortex of the brain. Leukocytes accumulate about the blood-vessels and nerve cells, particularly the motor ganglion-cells. Van Gehucten and Nelis have called attention to "the accumulation of lymphoid and endothelial cells around the nerve cells of the sympathetic and cerebro-spinal ganglia." In all probability various degenerations not now understood take place in the nerve cells though as yet it has not been established that other viscera have been affected. The pharynx and esophagus are congested and may become edematous. Pulmonary congestion is also a possibility. Hemorrhages from the mucous membranes may be detected and the perivascular spaces of the brain may show slight hemorrhagic extravasations while thrombi may be found in the cerebral vessels.

Symptoms.—The period of incubation varies from two weeks to three months, averaging six weeks. At the close of this period, rarely before, the premonitory symptoms appear. There may be slight irritation about the wound, the patient is depressed, irritable, sleepless and inclined to headache, loss of appetite and melancholia. There is always dread of some impending danger, and a bright light or sudden noise irritates the patient. Pains begin to radiate from the wound, the voice becomes husky, there is some difficulty in swallowing, there being at the same time a slight rise in the pulse and temperature. This stage usually lasts about twenty-four hours. The *stage of excitement* then ensues and is characterized by extreme excitability, restlessness and pronounced hyperesthesia. The patient is sensitive to the slightest noise or draft of air, the least disturbance of any kind exciting a violent reflex spasmodic contraction of the larynx. There is *extreme thirst* though an attempt to swallow water causes an intensely painful spasm of the muscles of deglutition and respiration. It is this spasm which arouses the patient's fear at the very sight of water and hence the term hydrophobia. The nervous excitement may become so extreme and the spasmodic contractions of the larynx may be so severe that *peculiar sounds* are emitted and a certain degree of *dyspnea* exists. The convulsive movements of the mouth are often so pronounced that the jaws snap together much like the vicious snappings of a dog, while the inability to swallow the increased secretion of saliva and its ejection from the mouth explains the laity's description of "frothing at the mouth." The *intervals* between the paroxysms are characterized by a degree of tranquility in which the patient appears quite normal. *Fever* is usually a feature of the paroxysms and the pulse becomes rapid and irregular. During the periods of extreme excitement the patient may become maniacal and the mental aberrations may develop in a state of melancholia or the patient may be, at times, inclined to suicide. This stage usually lasts only a few days.

The *paralytic* or concluding stage is characterized by a state of unconsciousness or coma. The spasms are no longer noticed and, in a few hours, death may intervene. A paralytic form of the disease may rarely exist without any signs of the stage of excitement. The duration of the disease is only about one week from the onset.

Diagnosis.—The hyperesthesia, the peculiar character of the reflex spasms with the difficult deglutition and the fear of water, together with the dyspnea and mental excitement with the history of a bite lead to a diagnosis of hydrophobia. *Tetanus* presents the closest similitude but this disease begins with trismus followed by opisthotonos while the period of incubation is always shorter. Also there are no laryngeal symptoms, there is no spasm in swallowing and the mental depression is absent. *Hysteria* should never be confused with this disease. A condition of *pseudophobia* or *lyssophobia* may be decidedly confusing, especially in case a patient has been bitten by a dog even though it does not have the rabies. This is associated with a neurosis or possibly hysteria and runs a longer course without any of the well-defined signs of hydrophobia.

Prognosis.—In a well defined case of hydrophobia which is thoroughly established a favorable prognosis is not to be expected. We may expect good results, however, from the preventive of Pasteur treatment and for this reason the deaths from this infection may be reduced to the minimum.

Treatment.—The *prophylaxis* must consider first of all the immediate cauterization of the wound after dog-bite for the purpose of destroying any germs which may have a systemic effect. Inasmuch as this is rarely done with sufficient promptness it is best to treat the wound as an infected one. The dog should always be kept under surveillance for the purpose of determining whether he has rabies. In case the infection is evident in the animal it should be promptly killed and a portion of the medulla should be obtained under strict asepsis. This should be put into a sterile bottle containing a solution of equal parts of pure glycerin and water that has been sterilized by boiling. The bottle should be forwarded to a Pasteur Institute where, by animal inoculation, the disease may be scientifically determined.

The *preventive inoculation* or Pasteur treatment is our only means at the present day of successfully combatting the disease. Pasteur ascertained that inoculations by virus from the brain, medulla and spinal cord of animals suffering with this disease could be utilized in the treatment of the same. Rabbits thus inoculated "produced a virus of such increased virulence that after twenty-five successive inoculations there resulted a virus that acted after a period of incubation of eight days; and after twenty-five additional inoculations in seven days. The virus from the medulla of rabbits, with this short period of incubation, is called 'fixed virus' as contrasted with 'street' virus. Now, although the spinal cords of such animals contain the virus in a state of great intensity, Pasteur ascertained that its intensity was greatly reduced by preserving the cords in dry air, and that it disappeared altogether in two weeks. The desiccation is practiced in sterilized glass vessels in which are placed pieces of caustic potash. If, now, dogs are inoculated with an emulsion of such medullas of reduced virus, say cords preserved for twelve or fifteen days, and then with the cord preserved for a shorter period—that is, with progressively stronger virus—they acquire immunity from the inoculation by the fresh cord of the rabid rabbit."

"Pasteur availed himself of these facts to inoculate human subjects who had been bitten. He used an emulsion of rabbit's cords that had been kept fourteen days, and on successive days made twelve more inoculations from cords preserved a shorter time, until those only one day old were used, after which immunity was secured." (Tyson.) Later Pasteur reported favorably on his so-called "intensive method" consisting in inoculations from cords of increasing virulence in more rapid succession. This is practically the method employed in the so-called Pasteur Institutes.

The *general care* of the patient will include the isolation from all noise and excitement while food must be administered by nutrient enemata. The spasms may sometimes be relieved by the inhalation of chloroform or by the internal administration of morphin or chloral.

TETANUS.

Tetanus or lock-jaw is an acute infectious disease caused by the tetanus bacillus and is characterized by paroxysms of tonic spasms which recur with increasing severity. Both human beings and some of the lower animals may be infected by this bacillus.

Etiology.—It is a disease which prevails more frequently in tropical climates and it is epidemic in certain parts of the West Indies. It may occur as an idiopathic disease or it may be the result of traumatism. In the new born it is known as *trismus neonatorum.* Punctured and contused wounds more frequently predispose than the incised wounds. The *bacteriology* of the disease has been established by the researches of Nicolaier, Rosenbach and Kitasato who demonstrated the presence of a specific organism, the bacillus of tetanus. This is a "long, slender rod at one end of which appears a swelling due to the formation of a spore in that locality, thus giving the organism an appearance like that of a pin or drum-stick." It is motile, develops at ordinary temperatures and is anaerobic. The bacilli do not enter the circulation, remaining at the site of the wound, and there develop a toxin which causes the symptoms. Brieger demonstrated the presence of three poisons, *tetanin, tetanotoxin* and *spasmotoxin,* which produce tonic convulsions thus establishing the toxic nature of the disease. The *infection* is developed by the entrance of the poison through a punctured or lacerated wound though it may occur idiopathically. The bacilli have a wide distribution, being found in the earth and putrefying matter and manure. *Immunity* has been established in animals by the injection of the cultures of the bacilli with the addition of iodin trichlorid which diminishes their strength. This serum has been successfully used as an antitoxin protection. In this country the Mulford Company has produced an antitetanic serum which is used in these cases.

Pathology.—No definite lesions have been found. The virus evidently acts upon the nerve cells of the medulla and cord, an inflammation or softening being noticed particularly in the latter. The nerves also show the results of the injury by their reddened and swollen condition, an ascending neuritis being frequently developed.

Symptoms.—The *incubation period* varies in the acute and chronic forms of the disease. In the former it continues for one or two weeks while in the latter it lasts two weeks or longer. *Acute tetanus* usually shows the prodromal signs of languor, headache, and some gastric disturbance for a short time before the real symptoms appear. *Muscle stiffness* is experienced in the back of the neck and the muscles of mastication. Very soon the tonic spasm of the masseters develops until a condition of *trismus* or *lock-jaw* exists. There may be an associate paralysis of the facial muscles, and there is often also an impairment of deglutition. Rigidity of the cervical muscles gives rise to retraction of the head. The *facial expression* is immobile, the eyebrows are raised, the forehead is wrinkled and the corners of the mouth are retracted giving the so-called "sardonic grin." In *children* the attack is liable to be limited to these parts. The tonic spasm, however, usually extends until other muscles of the body are affected. The muscles of the trunk may be rigid (orthotonos); or the muscles of the back may be affected (opisthotonos); flexion to one side is rarely noticed (pleurothotonos); and spasms of the abdominal muscles may cause the forward flexion (emprosthotonos). In *violent attacks* the thorax may be compressed, the respiration may thus be excited and spasm of the glottis may cause cyanosis. The paroxysms may last for some time with short intervals of relaxation, which is not always complete as the slightest irritation may cause their return. *Agonizing pain* attends the paroxysms and the patient seems to be held in a vise by the rigid contractions. There may be—in rare instances—a convulsive contraction of the diaphragm as the result of reflex irritation. The reflexes are usually exaggerated. *Profuse perspiration* is generally a constant symptom. The *temperature* is not often high though it may suddenly rise to 104° or 106° F. Just before death it may rise even as high as 109° or 110° F. The pulse is small, irregular and always rapid, being as high as 150 or 160 per minute. The *urine* may be suppressed or voided with difficulty on account of the muscular rigidity. Constipation often exists from the same cause. *Cephalic tetanus,* described by Rose, arises as the result of injury on one side of the head. It is characterized by stiffness of the jaw and paralysis of the facial muscles on the same side and difficult deglutition. Death may take place during the paroxysms as the result of heart paralysis or asphyxia or it may take place later following extreme exhaustion.

Chronic tetanus is characterized by manifestations similar to the acute form though they are not pronounced and develop more slowly. When the symptoms are aggravated there is liable to be a period of decided relief from the peculiar rigidity. These periods of relaxation are gradually prolonged until convalescence occurs. A relapse is always possible.

Diagnosis.—The trismus and muscular rigidity are usually sufficient to settle the diagnosis. As the spasms are similar to those of strychnia poisoning it is necessary to make a careful differentiation. In *strychnia poisoning* a history of its ingestion may be obtained, there is no sign of a local lesion, there is no period of incubation, gastric disturbance precedes the tetanic contraction of the extremities, violent convulsions also characterize the onset and there are intervals of complete relaxation. On examination the gastric contents reveal the presence of strychnia and the course is brief, death or recovery soon taking place. In addition to these symptoms the clearest point of difference is recognized in the lock-jaw of tetanus. In *tetany* the spasm affects the extremities, the hands and larynx being particularly affected, intermissions are common and its peculiar posture is distinctive. *Hydrophobia* is distinguished by the history of the bite of an animal, the reflex paroxysms of respiration, the psychic disturbances, the absence of lock-jaw, and the presence of dysphagia.

Prognosis.—In the traumatic cases at least 80 per cent are fatal and in idiopathic cases fully 50 per cent die. The mortality is not so great in children. A late appearance of trismus and the general rigidity, the absence of fever and the localization of the spasm to the muscles of neck and jaw may be regarded with some degree of hope.

Treatment.—*Local treatment* of the wound is of first importance. Disinfection and thorough cauterization of all penetrating wounds is demanded after free incision has exposed the bottom of the wound. Nitrate of silver is considered by many to be valuable as a germicide and for its cautery effect. Carbolic acid and iodin are also frequently used for this purpose. Some claim that amputation of the injured limb should be performed. *Absolute quiet* of the surroundings must be maintained, the patient being kept in a dark room where all excitement and noise are excluded. *Rectal feeding* is necessary on account of the lock-jaw though the nourishment may be given, in liquid form, by means of a catheter introduced through the nose. *Stimulation* by means of hypodermatic injections of strychnia and digitalis is of greatest importance to sustain the heart's action and to overcome the muscular contractions. *Inhalations* of *chloroform* during the spasms give much relief, and *morphin* (1/4 gr.) should be administered during the interparoxysmal stage for the relief of pain. *Chloral hydrate* (grs. 15 to 30), *potassium bromid* (grs. 15 to 20), *Calabar bean* (gr. 1/4 to 1/2), may be given from three to five times daily to adults. *Curare* (gr. 1/25) and cautiously increasing the dose has been recommended. *Gelsemium* tincture (gtts. 15 to 30) repeated until the physiological effect of the drug is obtained is highly lauded. *Tetanus-antitoxin* may prevent "further invasion of the tetanus toxin but it can not cure the infection that has reached the spinal cord and brain." This serum may be used (20 to 30 c. c.) for the first dose and 15 to 20 c. c. for the subsequent doses every five or ten hours after. A *hot bath* gives much relief to the spasms.

The *internal remedies* should be carefully considered according to their indications though as yet no marked results have been obtained by their use. Among these we should naturally think of *strychnia* first. It is possible that the continued use of the second or third decimal potencies may give some relief. It is best, however, to obtain the maximum influence of this drug by hypodermatic injection (gr. 1/60 to 1/20) repeated every three or four hours. *Aconite, cicuta virosa, belladonna, stramonium, passiflora, nicotinum, veratrum viride, hypericum, physostigma, picrotoxin, cannabis indica* and *conium* may be indicated.

PROBABLE INFECTIOUS DISEASES.

Muscular Rheumatism.—Muscular rheumatism is a term which has been erroneously used to define inconstant and inexplainable pains which appear from time to time in different parts of the body. The term myalgia is also used. At present it seems more probable that these migratory pains which are located in the muscles, their fasciae and the periosteum are either a residual condition following acute rheumatic fever, or a constitutional disease with local symptoms. The infectious cause is at least quite probable. Some regard the myalgic pains as a neuralgia of the sensory nerves which supply the muscles.

Pathology.—The pathology is not as yet well established. In fatal cases where post-mortem observations are possible, some swelling of the fibers, with slight granular changes, has been observed. When the disease is of long standing atrophy may follow as the result of trophic disturbance. The changes are similar to those of myositis, a round-cell infiltration of the connective tissue being found. If the disease is prolonged, there may be some swelling of the muscular fibers with slight secondary degeneration and the formation of vacuoles. Proliferation of the interfascicular connective-tissue may take place in cases of long standing.

Etiology.—The *rheumatic diathesis* has been claimed for a majority of these cases and there may be something in this so far as the soil conditions are concerned. We may also accept the theory of *hereditary influence. Exposure* also may predispose as a sudden suppression of the excretions or perversion of the circulation in a muscle may favor the reception of an infection or excite the inflammatory development of latent micro-organisms. *Previous attacks, sex,* so far as it pertains to occupation, and *age,* inasmuch as young adults are inclined to the acute and subacute forms, while older people suffer with the chronic form, may all be regarded in the light of predisposing or etiological factors.

Symptoms.—*Pain,* which at times is sharp, lancinating and paroxysmal or again deep-seated, dull and constant, is the most characteristic symptom. Changes in the weather, pressure and over-use of the muscles generally accentuate the pain, while the paroxysms also show a tendency to aggravations at night. *Fever* is rarely observed, though Leube claims the discovery of fever, together with slight cardiac murmurs, in about one-third of his cases. *Rheumatic nodules* may often be found in the larger muscles. The *duration* is short, though exacerbations or recurrences may take place when occupation and other predispositions favor its return.

Lumbago, or myalgia of the lumbar muscles, is a frequent form which the disease assumes. This is characterized by sudden onset and pain which is excited by stooping or a quick change of position which causes the muscles to contract. *Pleurodynia* is a term which is used when the intercostals, pectorals and serratus magnus are affected. Pain is then excited in case of sudden thoracic movement, or by the act of sneezing and coughing. *Torticollis* is a painful involvement of one or all of the neck muscles of one side, the head being drawn firmly or spasmodically to that side, thus relaxing the affected muscles. *Cephalodynia* is a general or local involvement of the head or scalp muscles, pain being felt when these are contracted or the scalp is touched. Other terms are employed when special muscles are affected.

Diagnosis.—The etiology, the pain which is always increased by contraction of the involved muscles, together with occasional slight fever, are the guiding diagnostic features. *Neuralgia* is differentiated by its painful points in comparison to the relief which is sometimes afforded in myalgia by pressure. *Dermato-myositis,* in addition to the presence of pain and swelling of the muscles, displays a redness and hyperesthesia of the skin.

Prognosis.—The prognosis is invariably good, a fatal result rarely following, though a patient may suffer from many recurrences.

Treatment.—If the disease is due to a previous acute attack of inflammatory rheumatism, as is frequently the case, the general treatment should conform to that which has been outlined under that subject. *External applications* of heat and muscle rest, obtained by complete immobilization of the inflamed parts are the first essentials of treatment. Hot fomentations, stimulating liniments, massage and sometimes electricity give both temporary and permanent relief. Dry heat obtained by modern appliances or by the application of hot water bags are valuable expedients and usually furnish good results. Camphorated oil or other oil rubbed into the affected part and then ironed in with a hot flatiron, the parts being well protected, is also efficacious. Covering of the affected muscles with warm flannels or the application of cotton held in place by a roller bandage, are measures which may also be considered. Counter-irritants, such as blisters, acupuncture and such severe agents which distract the patient are not now so generally used though they are of service in some cases.

The *internal remedies* must be selected according to their symptomatic suggestions though many features of specific importance should be considered. Any predisposing disease should receive attention. The general health of the patient should be improved and elimination should be encouraged in both acute and chronic rheumatism. The salicylate of sodium (grs. 10-15 q. d.) or potassium acetate in the same dosage will help any indicated remedy which may be demanded. The *diet* also requires strict attention and if there is any sign of fever, the food should be entirely of a light nutritious character. In case fever is absent, the patient should be well nourished, but the diet should not be too heavy. A vegetable diet is usually best, though a fair amount of fish and fowl may be permitted. The patient should be instructed to drink an abundance of water and, to attain the result desired, it should be prescribed at specific times during the day. *Palliation* may be attained by the hypodermatic or internal use of morphin (gr. 1/8 to 1/4) or codein and some of the coal tar preparations, in small doses, may be tolerated if there is no cardiac trouble.

The *indicated remedy* will be of most service and this is found only by a painstaking study of the case. *Bryonia* is naturally thought of first as it refers to that stitching and tearing pain which is aggravated by motion. It refers also to a redness of the joints and some swelling of the feet, while there is often an underlying digestive disturbance characterized by the well known symptoms of bryonia. *Sanguinaria* may be compared in many respects with bryonia, but it pertains more particularly to the pains of the head, neck and shoulders, motion always causing aggravation. *Mezereum* is frequently of value when the pain is in the neck or back or when it is deep-seated, the patient being extremely sensitive to the slightest touch. Any prolonged deepseated pain should suggest a specific cause, and thus *kali hydriodicum* and *mercurius* are always to be kept in mind. *Pulsatilla* meets many of the rheumatic symptoms when the pain is tense and shows a tendency to migrate from one joint to another or from one muscle to another. I have obtained good results from the use of the tincture. *Rhus tox* refers to toxic influence which has not been fully overcome. Swelling of the joint, pains in the tendons and deep fascia, the stiffness of the limbs · and the radiation of the pain along a nerve are characteristic symptoms of this remedy. It is especially valuable in lumbago. *Ledum* is a useful remedy when the pains attend a gouty condition and affect the smaller joints. *Colchicum* is always a standard agent either in acute or chronic rheumatic conditions and the symptoms of this remedy may be compared with those of *camphor, cocculus, colocynth, ranunculus* and *nux vomica*. *Phytolacca, cimicifuga, dulcamara, rhododendron* and *aurum muriaticum* are also frequently indicated remedies.

ACUTE MULTIPLE MYOSITIS.

This disease has also been defined as an infectious myositis or an acute poliomyositis. It is characterized as a primary acute or subacute inflammation of the voluntary muscles due to some unknown micro-organism. The inflammation invades the muscular fibers and to a certain extent the interstitial connective-tissue. Following the initial hyperemia there is an exudation of leukocytes and later the muscles become firm and fragile only to undergo a fatty degeneration after a time. Serous infiltration may take place later. The etiological factors and the predisposing influences are alike unknown.

Symptoms.—*Prodromal symptoms* such as headache, erythema, urticaria, herpes and splenic enlargement are frequently observed. *Fever* of moderate intensity is generally present and the pulse is proportionately accelerated. The *local symptoms* are characterized by painful sensations in the extremities and in the trunk which impair the normal functions. Pain is always experienced on pressure over the insertions of the muscles and there is some edema of the skin accompanied by transitory redness. The joints may also partake of the inflammatory invasion. In advanced stages the muscles of the pharynx and respiration may become involved, giving rise to dysphagia and respiratory embarrassment. Atrophy of the muscles may follow the inflammation and edema.

Diagnosis and Prognosis.—The disease is not easily recognized by the clinical picture just described. *Trichiniasis* presents a similar symptomatology, and is differentiated only by microscopic examination of a section of muscle. *Multiple neuritis* is attended by more pain, joint weakness, and no swelling or edema. Myositis may pursue a rapid course and terminate in two or three months or assume a chronic character and last for a long time.

Progressive Ossifying Myositis.—This is a rare disease, characterized by progressive calcification of one or more groups of muscles. It begins about puberty, affects males more often than females, and extends over years in its course. At first there is a low-grade inflammation which results in the deposition of calcareous plates of bony hardness in the muscles. Partial or complete ankylosis of the joints finally ensues. No case has ever been cured.

CHRONIC ARTICULAR RHEUMATISM.

The chronic rheumatic conditions are characterized by a slowly developing arthritic disturbance which usually follows an acute or subacute attack of rheumatic fever. The aged are particularly predisposed to this form of the disease and, to a certain extent, it is induced by occupation and exposure.

Pathology.—The joints show slight synovial inflammation and effusion with articular and periarticular thickening sufficient to cause stiffness and some joint deformity. There may also be slight cartilaginous erosion while muscular atrophy may follow in cases of long standing.

Symptoms.—*Pain* is usually the most prominent symptom and it is aggravated during sudden changes in the weather or following excessive use of the limbs. The joints may be slightly red and swollen, while tenderness and immobility are occasional symptoms. In many instances there may be a complete absence of joint changes. When the local symptoms are pronounced it is possible sometimes to elicit *slight crepitation* by manipulation. These cases usually develop more or less ankylosis in the course of the disease. The pain and stiffness is worse during the morning hours, but usually wears off later in the day after a certain amount of exercise. *Exacerbations* and *remissions* are characteristic phases of the local symptoms. *Constitutional symptoms,* which are dependent upon the chronic rheumatism, are rarely present. A marked general debility is usually incident to the disease and a certain degree of anemia may be present. Such patients usually suffer from neurasthenia and dyspepsia while chronic endocarditis is frequently a complication.

Prognosis.—A fatal result is not to be expected in these cases, though, appearing so frequently in advanced life, the disease may be a factor which favors a fatal intercurrent disease. A cure is rarely accomplished, though it is possible to palliate and, therefore, afford a more comfortable prolongation of life.

Treatment.—The *local treatment* will include joint protection and warmth by coverings of flannel and cotton and much relief is also obtained by the hot air treatment by special apparatus. Wet boracic acid dressings, well covered by oiled-silk and a bandage, is probably the best local treatment when there is any inflammatory excitement of the parts. The thermocautery has been employed in some cases and, in joint effusion, it is advisable to remove the fluid by aspiration. Massage and osteopathic manipulations are without doubt serviceable in many cases and particularly when muscular atrophy has developed. Electricity, both static and galvanic, will be of service in many cases of long standing and any tendency toward paralysis will surely suggest it. Painting the parts with iodin and the use of stimulating liniments, gives relief in many instances. *Hydrotherapy* is of much importance and it is generally best to send a patient to some of the springs where hot baths may be given and a good alkaline water can be obtained. The Hot Springs of Arkansas and Virginia are very good places for this treatment and many of the sanitaria are supplied with facilities for giving massage, baths, electric light baths and other forms of treatment.

The *diet* is a matter of much importance, for these patients need a thorough building up by increasing the nourishment and favoring free elimination. Milk, eggs, meats and farinaceous foods should be given in abundance. In many instances it may be advisable to encourage the appetite by the regular use of a good wine. So far as possible it is best to have the patient take a sufficient amount of exercise, lest with the increase of food, a gouty tendency be established. *Internal medicine* will be demanded according to indications, *iron, arsenic, strychnia,* the iodids and tonics are to be thought of.

WEIL'S DISEASE.

Weil's disease, or acute febrile jaundice, should be classed under the infectious rather than the hepatic diseases, as it is probably of specific origin. It is characterized by jaundice, remittent fever and muscular pains. The special organism is not positively known, though Jaeger claims it is due to the *bacillus proteus fluorescens.* It is more common in male adults between twenty and forty, the majority of cases occurring during the summer months. Butchers and brewers are said to be particularly predisposed.

Symptoms.—The disease develops suddenly with *fever* and usually chills, headache, vomiting, sometimes diarrhea and pronounced weakness. *Muscular pains,* increased by pressure, soon develop, while the temperature rapidly rises to 103° or 105° F. This remains high, with slight morning remissions, for three to six days, and the pulse is correspondingly accelerated. *Nervous symptoms,* with supervening delirium and somnolence, may appear, while *jaundice* of varying intensity may be observed any time between the third and sixth day, the stools often being clay-colored. With these symptoms hepatic and splenic enlargement are evidenced and nephritis is not uncommon. *Hemorrhage* may take place into the skin or from mucous membranes, while blood may appear in the vomit, sputum, urine and stools. Erythematous, herpetic and roseolar *exanthemata* are occasionally noticed. The *fever* remits during the course of the disease, which usually terminates by lysis in ten to fourteen days and convalescence begins. The muscles may remain sensitive and stiff for some time. In *fatal cases* the nervous symptoms and the hemorrhages are exaggerated and death may take place with coma. Autopsy in these cases has revealed "a parenchymatous degeneration and cellular infiltration of the liver and kidneys, enlargement of the spleen and hemorrhages in various organs."

Diagnosis and Prognosis.—The *diagnosis* rests upon the acute onset with fever, pains in the muscles, nephritis, and icterus. It is distinguished from *relapsing fever* by the absence of the spirilli in the blood. The *prognosis* as to life and recovery is fairly good, though a fatal end may be feared when the nervous symptoms are severe. The *treatment* is purely symptomatic and the remedies which are usually indicated have been mentioned under the diseases of the liver.

FEBRICULA.

Febricula or ephemeral fever is a simple continued fever which is of short duration and depends upon a variety of causes. It is characterized by a febrile attack which lasts only twenty-four hours or at the longest three or four days. It most frequently arises from digestive perversions due to the ingestion of improper food. It is frequently regarded as a gastric fever in children. Exposure, attendant diseases like tonsillitis and bronchitis, the action of putrid or decomposing foods and the inhalation of toxic gases may also be considered as etiological factors. The toxic products of such infectious diseases as rheumatism or malaria may also predispose to its development.

Symptoms.—*Fever* is the first noticed symptom and this has a sudden onset when it is ephemeral in character, while in other instances it may develop slowly. A *chill* or convulsions in children often attends a sudden rise of temperature. The fever is rarely higher than 102°-103° F. and terminates by crisis unless it assumes a subacute form. A *frequent pulse,* headache, nausea, loss of appetite, restlessness and lassitude are attending symptoms. The nervous symptoms may be pronounced and delirium may exist for a short time. The disease may assume several types, the cerebral, gastric or gastro-intestinal, which are characterized by the predominance of symptoms which point to a disturbance of these parts.

Diagnosis and Prognosis.—The *diagnosis* is usually made by eliminating other probable diseases such as the acute exanthemata, tonsillitis pneumonia and meningitis. *Typhoid* is frequently suspicioned and this is only settled by a prolonged observation and the later finding of the sero-reaction. In a majority of instances the fever subsides in a few days. Other differentiations are made in the early stages only by a painstaking study of the symptoms and the physical findings. The *prognosis* is invariably favorable.

Treatment.—These fevers usually respond to a careful limitation of the diet, free elimination, rest in bed and freedom from excitement. Hydrotherapy, and a mild laxative may decidedly shorten the attack. An intestinal antiseptic—particularly the sulphocarbolates—is of unusual value. The patient should always be isolated for a few days until we are assured that it is not a contagious disease. Such remedies as *aconite, belladonna, chamomilla, eupatorium perfoliatum* and *baptisia* are usually the only medicinal requirements for the fever. When gastro-intestinal disturbances are evident aside from the food limitations such remedies as *arsenicum, bryonia, ignatia, ipecac, iris versicolor, chelidonium, china* and *cuprum arsenicum, nux, antimonium crud.,* and *pulsatilla* may be demanded. When the nervous symptoms are marked *moschus, cimicifuga, sumbul, valerian* and *kali phos.* may be indicated.

MILK SICKNESS.

This is a peculiar infectious disease which afflicts both man and animals. In the latter it is known as "trembles." It prevailed extensively in the early settlement of our western frontier and it has always been traced to the eating of the flesh and drinking of the milk of diseased animals. It is now almost an extinct disease as it evidently disappears with the advance of civilization. The etiology and pathology are not fully understood. It is supposed that the poison is derived from the soil and Phillips claims to have discovered a spirillum in the blood. "When a cow is very ill, food is refused, the eyes are injected, the animal staggers, the entire muscular system trembles and death occurs in convulsions sometimes with great suddenness." (Osler.)

Symptoms.—In man the symptoms appear after a short *incubation period* in which restlessness and general distress are followed by gastric pains, nausea and vomiting, fever and intense thirst. The breath emits a peculiar odor and osbtinate constipation usually exists. The tongue is swollen and tremulous, while the nervous symptoms are marked by an extreme restlessness which may merge into mental dulness simulating that of typhoid or a state of coma and convulsions may ensue. In some cases the temperature is elevated while in others it may be subnormal. The *duration* of the disease is variable. It may continue for a week or ten days, or in the distinctly acute form, death may occur inside of three days. The *prognosis,* in many instances, is favorable, though death due to asthenia may suddenly take place. Osler states that an ounce of butter or cheese, or four ounces of the diseased beef, given three times a day, will kill a dog within six days.

Treatment.—Aside from the symptomatic treatment the profession knows but little regarding the management of this disease. The diet should be limited and stimulants should be administered and free elimination is to be encouraged. Prophylaxis should consider a careful exclusion of the milk and flesh of affected animals.

MILIARY DISEASE.

This, known also as sweating sickness, is an infectious disease of rare occurrence, which is characterized by copious perspiration and a vesicular or miliary eruption. It has prevailed epidemically in many foreign countries. but has not been commonly observed in America. The exciting cause is unknown and the pathology is not fully understood. It is supposed to occur most frequently in the spring and summer months and those of late adult life are more often affected.

Symptoms.—A *fever* with "copious and persistent sweats," is a prominent symptom. On the third or fourth day an *eruption* attends the sweating and this is characterized by its miliary vesicles. These burst and within a day or two a scaly desquamation succeeds the eruption. *Gastric disturbances* are prominent and, in severe cases, grave delirium may intervene. *Hemorrhages* may occur and give rise to a fatal collapse.

The *prognosis* is not fully settled, the disease being so rare that there has been slight opportunity to study the pathology and symptomatology. In its epidemic occurrence the fatalities have amounted to about ten per cent. Serious relapses have been known to occur.

Treatment.—*Quinin* and *atropin* are the only two special remedies which have so far been recommended. It would seem probable that such homeopathic remedies as *eupatorium, belladonna, veratrum alb., rhus tox, atropin, camphor* and *agaricin* would naturally be indicated.

FOOT AND MOUTH DISEASE.

This is an acute infection of cattle, sheep and pigs which spreads with great rapidity. The micro-organism has not yet been discovered. In animals, after a short incubation period, the fever develops accompanied by swelling of the oral mucosa on which small vesicles soon form. These contain a clear fluid and soon rupture, forming small ulcers. Ptyalism is pronounced and general asthenia is marked. The furrows of the animal's feet and the udders may be similarly affected, while the milk becomes yellow-white in color and has a bitter nauseating taste. It is from these epidemics among cattle that the disease is conveyed to man by the milk of the animal, it being generally characterized as an *epidemic stomatitis,* or *aphthous fever.* It is possible that the disease may also be communicated by the butter and cheese made from the infected animal. Boiling of the milk destroys the virus. The disease is rarely conveyed by inoculations obtained by milking, but the flesh of the animal apparently does not cause the infection.

Symptoms.—There is a short *incubation period,* lasting only two to five days. A *rigor* or slight chill may precede the fever which is not high. The *vesicles* and aphthous condition are soon recognized on the predisposed parts, the mouth being generally very hot, red and swollen, while salivation is often marked. The integument, in rare instances, suffers from a miliary eruption which may advance to the pustular stage. Hemorrhages are exceptional but serious symptoms.

Diagnosis.—The prevalence of an epidemic will direct attention to the typical symptoms, as otherwise the miliary eruption would suggest other eruptive diseases. The *prognosis* is usually favorable, unless there should be rare complications. The *treatment* will pertain to the use of indicated remedies which have been mentioned under the subject of Aphthous Stomatitis. Prophylaxis is obtained by avoiding the use of milk from suspicious cattle.

GLANDULAR FEVER.

Glandular fever is a term used to define an infectious disease of children which is characterized by slight redness of the throat, high fever and lymphadenitis involving especially the anterior cervical groups. The anterior cervical lymphatics are first affected, the affection starting in the tonsils or pharyngeal mucous membrane. The glandular enlargement may spread to the inguinal and the axillary glands. No special causative micro-organism has yet been discovered. The disease usually prevails among children between the ages of one and twelve years. epidemics occasionally taking place.

Symptoms.—After an *incubation period* of four to six days there is a sudden onset which is characterized by pain on moving the head and slight swelling on the neck. Nausea, vomiting and abdominal pain are also attending symptoms. The throat is sore, the tonsillar and pharyngeal mucosae are injected, there are slight nervous symptoms and the temperature vacillates between 101° and 103°. The *glandular enlargement* is noticed first on one side, but later both sides are affected. They are painful to the touch, but there is only slight redness and swelling, though deglutition is somewhat painful. Should the bronchial and tracheal glands become involved a troublesome cough and dyspnea may ensue. Bronchitis, hemorrhagic nephritis and otitis media are occasional complications. Suppuration of the glands rarely takes place and the swelling invariably disappears after a short duration.

Diagnosis and Prognosis.—It will be necessary to differentiate the swellings of glandular fever from those which may attend an attack of influenza or tonsillitis.

Treatment.—A clean intestinal tract with free action of skin and kidneys will help to cut short the attack. A nourishing but easily digested diet is indicated. *Belladonna, phytolacca, mercurius biniodid, arsenicum iodid, baryta iodid, echinacea strychnia ars.,* and *china ars.,* will be of great service. Cold or hot compresses to the neck, or the use of some of the plastic dressings will also add to the patient's comfort and aid in reduction of the inflammation.

CONSTITUTIONAL DISEASES.

DIABETES MELLITUS.

This disease is now regarded as a disorder of nutrition in which sugar accumulates in the blood and is excreted in the urine. It is clinically associated with polyuria and progressive loss of strength and flesh. The pathogenesis of the disease is still undetermined and many theories as to its cause have been deduced. It is, however, no longer considered to be a renal disease.

The most plausible theory of today is to the effect that it is dependent upon some organic or functional *disease of the pancreas* and this has an apparent confirmation in the fact that extirpation or loss of function of this gland are followed by glycosuria. It is claimed that the pancreas, or rather the cells in the islands of Langerhans, supply an internal secretion which contains a glycolytic ferment. This ferment is similar to that in the blood which permits the assimilation of glycogen. When organic disease or functional impairment of the pancreas interrupts this secretion, sugar will appear in the urine. Dr. A. C. Croftan holds that the origin of the blood-ferment is in the leukocytes and that this, with the pancreatic ferment, is identical with trypsin. In refutation of the pancreatic theory, we are obliged to admit, plausible as this may appear to be, that many cases of diabetes have existed in which no pancreatic disorder has been found post mortem. It, therefore, leads us to infer that other pathologic factors must evidently be associated with this in order to establish a clear case of diabetes.

Lesions of the nervous system have, for a long time, been regarded as the cause of this disease. This deduction was primarily established by the fact that irritation of the floor of the fourth ventricle of the brain produces polyuria and in some instances, at least, glycosuria. Tumors and abscesses of the brain which create pressure in this vicinity have likewise given rise to diabetes. It has been furthermore discovered that *injuries of the spinal cord* and more particularly disturbances of the sympathetic nervous system, have also been associated with the appearance of sugar in the urine. Some students of this subject have even gone so far as to claim that the disease is due entirely to the perversion of the sympathetics which govern the vasomotor function, and others have further claimed that it is a disturbance of the sympathetic nerves in the vicinity of the pancreas which alters the function of that organ and inhibits the action of the glycolytic ferment.

Interference with the *glycogenic function of the liver* is also considered as a possible cause of diabetes. This might naturally arise from hepatic diseases, or result from vasomotor stimulation and consequent hepatic congestion. Section of the pneumogastric is followed by vasomotor paralysis in which glycogen disappears from the liver and appears in the urine. An *alimentary glycosuria* has been described "as the result of the ingestion of more carbohydrates and peptone than can be stored up in the liver as glycogen, so that some of the latter finds its way into the hepatic vessels with resulting glycosuria." (Anders.)

The *relation of the suprarenal gland* to glycosuria has been given much attention of late. It was first established that glycosuria might be caused in animals by the subcutaneous injection of a freshly prepared suprarenal extract even when the diet was free from carbohydrates. Croftan deduced from this the theory that the suprarenal gland contained a substance which either caused the formation of sugar or inhibited its normal destruction. Herter does not accept this conclusion, but claims that diabetes exists when there is an interference with the normal oxidation in the cells of the pancreas. By a series of experiments with subcutaneous injections of adrenalin chlorid he formed the conclusion that a deranged suprarenal function interfered with the normal pancreatic function and thus this suprarenal perversion was one factor in a pancreatic disturbance sufficient to create diabetes. In line with this reasoning it may be truly stated that any other influence (whether it be organic, functional, traumatic or neurotic in character) which directly or indirectly perverts the normal cell activity of the pancreas may lead to diabetes.

Many other theories have been advanced, but none as yet has seemed quite as tenable as those pertaining to the pancreas. The microbic theory has not yet been established, though many efforts have been made to find a definite micro-organism. Pavy's view, as described by Anders, is that "the carbohydrates of the food are converted into fat by the protoplasmic action of cells in the intestinal villi and enter the system in the same way as do fats taken as such. The surplus carbohydrates that escape the action of the cells of the villi are transmuted into glycogen in the liver. The glycogen stored in the liver obviously forms fat also, since this organ has some fat-forming function, and there are thus two barriers preventing the carbohydrate matter from entering into the blood and if either is deranged hyperglycemia with the consequent glycosuria results." We shall probably never know the real cause of diabetes until we understand the principles of perverted metabolism.

Etiology.—In considering the etiology it will be necessary to give some thought to the fact that diabetes may apparently exist without any relation to other diseases. On the other hand we may associate it directly with pancreatic, hepatic, brain or spinal cord diseases. *Traumatism* is also a prominent factor in its development, the records showing a preponderance of cases which develop soon after its occurrence. *Heredity* seems to have a decided influence as it may frequently be traced back in the family history. The Hebrew race seems peculiarly predisposed, while the colored race is apparently exempt. Swedes and Italians are the nationalities which also furnish many cases. *Males* evidently suffer more frequently than females, though statistics are unreliable in this respect. *Middle or advanced life* is the age at which it prevails, though it may exist in infancy. *Temperament* has much to do with its development, as it seems to occur most commonly with those of marked neurotic tendencies. Those in the fashionable class who are neurasthenic are frequently predisposed. Nervous shock, mental strain or nerve exhaustion may be the inciting influence. *Chronic diseases* like syphilis, malaria. and gout have been counted as causes. *Obesity* without doubt is a factor to be considered, as we frequently find diabetes in fat men of sedentary habits. *Pregnancy* is many times regarded as a cause, and diabetes may also follow the *infectious diseases.*

Pathology.—The morbid anatomy which pertains to diabetes must be looked for in the organs which are apparently concerned in its development. The *liver,* which has such a definite relation to the glycogenic function of the body, presents many changes. In the early stages of the disease the organ is hyperemic, the acini are enlarged and the capillaries are dilated. The liver cells are also enlarged, nucleated and somewhat fused. The minute changes, according to Rindfleisch, are more marked in the peripheral zone of the lobule than in that of the portal vein; the intermediate zone is fatty while the central part is quite normal. Hypertrophic and atrophic cirrhosis may both be found, but the organic hepatic diseases are not usually associated with diabetes.

The *pancreas* generally displays marked changes and these pertain to a lesion affecting the islands of Langerhans. As the cells in this region are the source of the internal pancreatic secretion which controls the glycolytic ferment, they often present some evidence of degeneration. The *kidneys,* though not affected primarily, become hyperemic as the result of constant irritation from the elimination of sugar and the signs of a catarrhal nephritis may be observed. In exceptional cases, as the disease advances, fatty or granular changes may develop. The *lungs* may become involved and the changes incident to bronchitis and pneumonia may be present. Tuberculous cavities and softening are not uncommon, causing the so-called diabetic phthisis. The *heart* is not often involved, though in rare instances arteriosclerosis with hypertrophy may be found.

The *nervous system* may be the seat of many perversions. The brain may present areas of congestion, extravasation, softening or sclerosis, while a tumor near the floor of the fourth ventricle or pressing upon the vagus and other cranial nerves is not an uncommon pathologic feature. Variations in the normal structure of the posterior part of the spinal cord, and peripheral neuritis are sometimes noticed. "Diabetic tabes" is a condition dependent on a multiple neuritis. Enlargement and sclerosis of the sympathetic ganglia are occasionally discovered. The *stomach* and *intestines* may present marked catarrhal changes. The lower *urinary tract* may be irritated by the constant passage of saccharine urine and this inflammation often extends to the external genitals causing a severe pruritus. The *blood* shows an increased percentage of sugar, though this has no apparent relation to the sugar in the urine.

Symptoms.—The disease may be recognized in its *acute* and *chronic form,* though the symptomatology varies only in the rapidity of development. The acute variety is of rare occurrence and is observed most frequently in young patients, the emaciation appearing early and to a marked degree. Diabetes, however, is recognized principally in its chronic manifestations. It is possible also to divide the cases according to the lipogenic or dietetic conditions, in which we observe the glycosuria of fleshy people, or the neurotic cases, which refer to the perversions of the nervous system, or again, that variety of cases which relate directly to a pancreatic disorder. As the symptomatology, however, is essentially the same in each instance, we may safely give the salient features as belonging to all varieties.

The *onset* of the disease, in the typical case, is gradual and the first symptoms which cause complaint are *polyuria and thirst.* The amount of urine may vary from four or five pints to as many quarts. The specific gravity is high, and sugar, varying from a fraction of one per cent to five or ten per cent, may be detected by urinary analysis. These two features will often present a wide disparity since the urine may sometimes contain sugar and still have a low specific gravity. To establish the true diabetic condition the presence of sugar in the urine must be constant and persistent. Sugar will always be increased in its elimination after a meal so a fair comparison of the twenty-four hour urine must always be made. In *appearance* the urine is usually frothy on account of its increased density and decidedly pale in color, though it may become cloudy after standing for a while. The odor is normal or sometimes sweet in fresh urine, but in the later stages it takes on an acetous odor from the presence of acetone and diacetic acid.

Urea is generally increased as the result of tissue destruction or possibly from an excess of nitrogenous food in the diet. Uric acid is normal or slightly increased, ammonia is often greatly increased and the same is true of phosphoric acid, lime and creatinin. *Albumin* is often present, but does not necessarily indicate the coexistence of a marked nephritic complication. Acetone diacetic acid and β-oxybutyric acid are frequently found in diabetic urine.

Dryness of the skin and itching are marked symptoms from the beginning. Boils and carbuncles are of frequent occurrence as the result of malnutrition and infection. Eczema, with terrible itching, is not an uncommon feature and labial and genital pruritus are often sources of great disturbance. Gangrene, particularly of the toes, may appear and is often due to slight traumatism. In conjunction with the dryness of the skin extreme thirst and frequent urination, we shall invariably discover many perversions of the digestive apparatus. The *appetite* is abnormally large and there is frequently an insatiable craving for carbohydrates. Dyspeptic symptoms are therefore common and constipation may be exceedingly troublesome. The *tongue is dry,* rough and fissured; the saliva is scanty and may give the sugar reaction. The teeth are inclined to decay early and aphthous stomatitis or thrush may develop on the mucous membrane of the mouth. The breath may have a peculiar acetous odor due, no doubt, to the acetone and diacetic acid in the blood. The liver is slightly enlarged though there is no great disturbance of the biliary function. Hypertrophic cirrhosis may develop in the later stages of the disease when an aged person is affected and with this there may be slight ascites, together with splenic enlargement. Icterus is not usually a marked symptom though the urine contains bile pigment.

The *respiratory symptoms* will pertain to pneumonic, bronchial and tuberculous conditions. Phthisis is always to be feared in every case of diabetes. It is not uncommon to detect a troublesome cough among the earliest symptoms. The *nervous symptoms* are very prominent. *Diabetic coma* is the symptom of speedy termination in death. It occurs more frequently in the advanced stages of diabetes and is characterized by a sudden or gradual unconsciousness; convulsions are rare, the eyes are half-opened, the pulse is feeble and frequent, the inspirations are deep, the expirations are short, and cyanosis gradually develops. The temperature at first is slightly elevated, but soon it becomes subnormal and death usually intervenes in twenty-four to forty-eight hours. Abortive forms of coma may appear during the earlier stages of the disease; these may end in apparent improvement, only to be followed by an unexpected attack which terminates in sudden death. In some instances it is brought on by exhaustion, over-exercise or in association with the symptoms of intoxication. The

√

cause of diabetic coma is not yet known, though β-oxybutyric acid (or its derivative acetone) is commonly held to be the exciting cause.

Peripheral neuritis is occasionally a complication; it is most frequently defined by a condition of "diabetic tabes," in which it will be possible to detect the peculiar ataxic gait, lost knee reflexes and the fulgurating pains. Other symptoms of neuritis, such as trophic disturbances, in which there may be a perforating ulcer of the foot and shedding of the nails together with the usual paresthesia, are occasionally observed. Irritability and hypochondriasis are common mental symptoms. The special sense disturbances may include the formation of cataract resulting in blindness. Other visual defects such as myopia, amblyopia and loss of accommodation are not infrequently found. Ophthalmoscopic examination may reveal a dilatation of the retinal vessels with hemorrhagic and inflammatory developments which may end in optic atrophy. Impairment of hearing, roaring in the ears and perversions of taste and smell are also symptoms of further special sense derangement. Sexual involvement may be manifested by early impotence.

Muscular symptoms may be recognized by the occurrence of cramps in the calves of the legs, with marked aggravations at night or in the morning. Similar disturbances of the abdominal muscles may be determined by gastric crises in which some epigastric pain is followed by vomiting and slight fever. The *heart* is liable to undergo hypertrophy with secondary dilatation. Atrophy of the cardiac muscle or fatty infiltration, with a subsequent dilatation of the cavities, are more common results of the constantly increasing debility. Feeble heart action is, therefore, always to be feared in the later stages of the disease. The pulse may, for a time, be quite normal or again it may be slow, accelerated or arrythmic; the tension may vary in accordance with the attending arteriosclerosis which frequently exists. Dyspnea with syncope may be looked for at any time under these pathologic conditions. The progressive loss of flesh and strength is always indicative of a fatal termination.

Diagnosis.—The diagnosis of diabetes should not be a difficult matter when the symptoms just outlined are carefully comprehended. The gradual and yet persistent loss of flesh and strength, the great thirst, and dryness of the skin, the polyuria, persistent glycosuria and later acetonuria, the inordinate appetite, the cutaneous symptoms and the other complications mentioned should be sufficient evidence. The confirmation obtained by urinalysis will absolutely settle the diagnosis. This should be performed by those who are able to give an expert opinion. Casual examinations of the urine by busy practicians are often misleading. Such tests as Fehling's, Trommer's and Haines' will show the presence of even minute quantities of sugar. Quantitive determination will be demanded under all circumstances and for this purpose the *fermentation test* is best. In this the specific gravity is taken before and after fermentation and the difference will indicate the number of grains of sugar in each fluid ounce of urine. If, for instance, the specific gravity before fermentation is 1.045 and after fermentation 1.035, the amount of sugar will be ten grains to the fluid ounce, each degree of loss in specific gravity representing one grain of sugar to the ounce of urine. The percentage of sugar may be determined by multiplying the decimal in specific gravity by 0.23. Legal's test for acetone, diacetic acid and oxybutyric acid will be very satisfactory.

Prognosis.—It is impossible to give an opinion regarding the outcome of this disease which will apply in all cases. Age is a factor for consideration. When the disease appears in the young it generally pursues an acute and unfavorable course. In every case we should be guarded in our prognosis as the depletion, following the loss of sugar, is inclined to increase until the strength is exhausted and the heart can not endure the strain. In subjects past middle life who are fleshy and disposed to gout the disease may be controlled if the treatment is prompt and thorough. In these cases the patient may live many years if the proper dietetic regimen is instituted. The thin, nervous, exhausted and overworked patients will not respond so readily to treatment and in such cases the prognosis is always unfavorable. Other complications, gangrene, nephritis, phthisis and particularly the appearance of coma, will be indicative of an unfavorable termination.

Treatment.—The treatment should be considered under three distinct heads, the dietetic, the hygienic and the medicinal. On general principles it may be stated that the *diet* should be so regulated that as far as practical all foods containing starch or sugar will be excluded. This will reduce the diet to a nitrogenous basis with an absolute elimination—if that is always possible—of carbohydrates. In certain cases departure from this principle is often necessary. This is discussed in a succeeding paragraph. A strict antidiabetic dietary includes fresh and salted meats of all kinds; soups made from meat, but without flour; game, oysters, crabs, lobsters and eggs are generally allowed. Green vegetables may be used freely and some claim that cream, curds, milk and buttermilk may be prescribed with safety. Sweet milk and cream, however, may better be supplanted by buttermilk. Fats are generally tolerated and fruits or nuts may be used with some restriction, but when the ingestion of fats increases the amount of acetone and fatty acids in the urine it should be restricted. Tenné, however, says that fat is not to be regarded as the source of oxybutyric acid as many cases continue for a long time consuming large quantities of fat without the appearance of these acids in the urine, and the diabetic coma is liable to attack those individuals who have lost their fat rather than those who are obese. Bread must be avoided unless it is strictly of the glutenous character. The following, quoted from the Practical Medicine Series for 1905 merits serious consideration.

"C. von Noorden reported to the meeting of Naturalists at Carlsbad in 1902 a number of cases in which the excretion of sugar continued upon the strict antidiabetic diet, but which were sugar-free when they received a large amount of oatmeal along with some vegetable proteid or white of egg and butter, other carbohydrates and meat being excluded. With the diminution of sugar the excretion of acetone and similar bodies together with ammonia was markedly reduced. Upon return to the ordinary diet great caution must be exercised lest the amount of the acetone bodies rise to an undesirable height. The author uses Knorr's oatmeal or Hohenlohe's oat-flakes. It is cooked for a long time with water and some salt; during the cooking, butter and a vegetable albumin, or after cooking, beaten white of egg, are added. The ordinary daily amount is 250 gm. of oatmeal, 100 gm. of albumin and 300 gm. of butter. The soup made in this way was taken every two hours. In addition some cognac, or wine, or some black strong coffee, was frequently given. The treatment has been used in over one hundred cases with various results, but in most cases favorable ones." The editor then adds that he "has used the von Noorden 'oatmeal' treatment in several cases, seven of these over a considerable period of time. Of the seven, two became worse with the diet and showed marked acidosis. Five improved very much, gaining in weight and vigor. The sugar of the urine diminished or disappeared, as did acetone and diacetic acid. The patients did not dislike or tire of the diet until it was continued for two or more weeks. When used for two weeks, and then replaced by an ordinary diabetic diet for two weeks, there was less objection. The diet is especially valuable as a means of testing the digestive and sugar destroying power of the individual."

Quoting further from the Practical Medicine Series: "There appear to be two classes of diabetics: One class bear the deprivation of carbohydrates badly, but when they are allowed carbohydrates of any kind excrete at first more sugar than before. After a few days, however, the excretion diminishes and in spite of greatly increased ingestion the excretion may continue to diminish until they are entirely free from sugar. These may be completely cured unless, as too often happens, they throw caution to the winds and indulge so freely as to bring on a return of the malady. In these cases it does not appear to matter what kind or kinds of carbohydrates are used. In the other class the source of carbohydrate appears to be of importance, and although the patients do badly upon a diet deprived of carbohydrates altogether and also upon one consisting of a mixture of carbohydrates. they do well when put upon a single food containing carbohydrates. In this case it is not sufficient that the carbohydrates should be chemically identical, but the exact botannical source appears to decide whether it will be well borne or not. If oatmeal is mixed with wheat bread the results are either unfavorable or lacking altogether, although both contain the same kind of carbohydrate, viz., starch. If a patient has shown himself to tolerate 250 gm. of oatmeal it can not be concluded that he will bear 250 gm. of wheatbread; on the contrary, experience shows that he will frequently excrete sugar as soon as the wheat bread is substituted for the oatmeal, and this goes on in increasing amounts from day to day. It is possible that there are differences in the starch derived from different grains so that one can not replace the other, or it may be that other constituents of the different grains cause a different reaction to the starch."

Beverages like tea and coffee, without sugar, may be permitted with careful restriction as to amount. Sweet wines and malt liquors must be avoided and, in my opinion no alcoholic drinks should be allowed; some writers, however, recommend the moderate use of alcohol which helps the patient to eat fat more easily, and it is itself a food because it is burned up in the system. Water is the best beverage and this should be used freely. Many special springs have been commended for their curative waters in this respect, but pure spring water of any kind will often suffice. Vichy, Seltzer, Appolinaris, Allouez, Waukesha, and many of the lithia waters are serviceable in this respect. In following the dietetic principles it will be well to refer to many of the modern diet lists found in the accepted text-books. Under all circumstances it is wise to give the patient a list of foods which may be adhered to, with special enumeration of those which must be avoided.

Under the *hygienic* considerations we must look to the care of the body and all its functions so as to institute a normal condition so far as possible. Bathing is of paramount importance and, for those who can endure it, a cold bath or sponge should be resorted to daily. The body should be protected by a sufficient amount of clothing, though it is not necessary to overburden a patient in this respect. Some need flannels to insure a proper amount of body heat, while to others excessive clothing is only a source of discomfort. Hygienic care necessarily includes moderate and regular physical exercise sufficient to establish a perfect circulation and at the same time to aid metabolism. Ventilation of the living and sleeping apartments will also require careful attention. Mental excitement, worry and nervous exhaustion must be obviated. Rest will demand equal attention with exercise in every case. Climatic change is often essential, the equable and temperate climate being most favorable to recovery.

The *medicinal treatment* has not as yet attained satisfactory results, though it seems unnatural not to hope for help in this respect. While I am unable to give positive assurance as to the value of internal remedies in this disease, I am equally confident of their adjuvant if not specific value. In the early medicinal treatment much confidence was placed in *opium* under the theory that it exerted a favorable influence over the polyuria and the excretion of sugar. It was used, in the early stages, in one grain doses and this was gradually increased to five grains two or three times daily. *Morphin,* in quarter-grain and increasing doses, or *codein* in similar dosage were often substituted for opium. The relief of many of the symptoms, particularly thirst, and nervous excitement, were helped by this method, but few in modern practice will claim any permanent benefit from these agents. *Arsenic,* after opium, has received most attention and it is highly probable that we shall hold to this remedy with a fair degree of satisfaction. Its action upon the gastro-intestinal tract and on the red blood corpuscles no doubt has a restricting influence upon the conditions which favor diabetes. Fowler's solution, beginning with small doses and gradually increased to its physiological action, is a popular remedy with many practicians. The *liquor bromid of arsenic* (2-5 drop doses three times daily), or *arsenate of iron* (gr. 1/16-1/6), has also been recommended. The *bromid of arsenic and gold* mixture which is known as "arsenauro," has been highly lauded for the same purpose. *Bromid of potash* (grs. 10-20), three times daily, has been advised in cases with marked nervous manifestations. *Sodium salicylate* (grs. 15-20) has also been employed when a lithemic history was present. It is claimed that, in such instances, the excreted sugar will be decidedly diminished by this agent. *Aspirin* may be substituted for this.

Guaiacol, quinin, salol, benzosol, methylene blue, antipyrin and *jambul* have been used with various claims for their efficiency. With the exception of the latter little can be said in their favor. The extract of jambul (grs. 10-20) three times daily often yields good results. Coal tar derivatives like *antipyrin, antifebrin* and *phenacetin* have been resorted to, but there is no special indication for their use and they should not be prescribed unless for the relief of pain with attending fever, and only then with proper protection for the heart. *Inhalations of oxygen* have been favored and *lactic acid* has been advised in heroic doses. *Cod liver oil* is considered by Tyson as a useful remedy when the carbohydrates are totally converted into sugar and eliminated, while the body albumin is being encroached upon. *Antisyphilitic treatment,* by means of iodid of potash and mercurial inunctions is probably serviceable in cases where syphilis is suspected. *Uranium nitrate* is a standard remedy which is probably one of our best therapeutic agents today. The dose is one to three grains and this may be gradually increased; it is best given after meals and well diluted with water. The acetate, ten to fifteen grains, is preferred by some. The *alkalies,* especially the alkaline carbonates, have been favorably employed for a long time. Potassium and sodium carbonate (grs. 10-20) may be given three or four times daily. The alkaline waters may be used much more freely and the results are probably fully as satisfactory. *Pancreatic preparations* have been employed in recent practice. These are given as the dry or glycerin extracts and without doubt much relief has been obtained, so far as the pancreatic perversion is concerned, by this method of treatment. *Thyroid extract* and *extracts of the liver,* as well as *suprarenal extracts,* are being investigated with the hope of palliation inasmuch as they, with the possible exception of the thyroid extract, apply to the etiological theories.

Special therapeutic suggestions will pertain to the symptomatic treatment and the complications as they may arise. *Pain* may demand an opiate or an anodyne, though Mitchell claims that it may generally be relieved by *lithium benzoate* (grs. 2-10) frequently repeated. Gastric disturbances may call for digestive assistants like *pepsin, pancreatin, hydrochloric acid, taka-diastase, caroid, sodium bicarbonate* or *subnitrate of bismuth.* Constipation will frequently demand special attention, but the saline waters and the usually employed mild cathartics will ordinarily answer for this purpose. Insomnia and debility will require the remedies indicated for these conditions, and such additional adjuvants as *phospho-albumen, phosphagon, phospho-glycerate of lime* and *lecithin.* Thirst may be relieved by bits of ice or, better, by hot alkaline mineral waters prescribed ad libitum. Itching of the skin will respond, temporarily at least, to warm baths, frequent sponging or the application of non-irritating dusting powders. The regulation of alimentary perversions will help this very much. Pruritus and eczema—particularly of the orifices—will require the application of *boracic acid solutions* and the careful drying of the parts. *Ichthyol, aristol* and *tumenol cerates* may be used to obtain relief. *Diabetic coma* will require prompt and vigorous attention. Alkaline waters must be crowded and rectal, subcutaneous or intravenous injections of normal salt solution must be employed early and persistently. Catharsis and sweating are also recommended in this emergency and the heart will need stimulation by digitalis until diuretic effect is obtained. To obviate diabetic coma the physician should constantly watch for the appearance of diacetic or oxybutyric acid and acetone in the urine. This condition will indicate the necessity of changing the diet, and at the same time prepare for the onset of coma.

Before entering upon the discussion of the remedies which pertain to specific indications, I will report a method of treatment which has been unusually successful in my work during the past two years. For this purpose I will select three cases for illustration. One was a man beyond sixty who was passing 6 per cent of sugar and voiding over 4,000 cc. of urine in twenty-four hours; he suffered also with marked gangrene in the right toe. The next case was a man past forty who had made his annual pilgrimage to Carlsbad for two or three years with only slight improvement from this and dietetic care. The last case was a young woman under thirty who had suffered with diabetes for about a year. All of these cases were typical and require no further description. The first case, after six months' treatment, is voiding only 1,600 cc. of urine in twenty-four hours and sugar amounts to only 0.6 per cent, while the gangrenous toe has been amputated with a perfect healing of the parts. The two other cases are eliminating no sugar in the urine and all are improving in strength and flesh. Aside from dietetic considerations in each case, I have employed the following treatment: *Tryptogen* (a tablet containing trypsin, ptyalin, amylopsin and bromid of gold (gr. 1/100) and arsenic (gr. 1/200) two to five tablets after meals, and *syzigium,* first decimal potency, four grains six times daily.

The homeopathic remedies with their suggestive symptoms are given as follows: *Arsenicum* refers to the insatiable hunger and the unquenchable thirst, though neither food nor cold water are tolerated by the stomach. It is also indicated when cachexia, anemia and skin eruptions, with intense burning and itching, are in evidence. The patient is also restless, anxious and exhausted; polyuria is marked and watery diarrhea is a frequent complaint. *Phosphoric acid* is naturally to be thought of in connection with any debility from nervous causes; polyuria is its chief symptom and sexual debility will also be a characteristic indication. This may be compared with *acetic acid, physostigma, phosphorus, picric acid, ammonium mur.* and the *arsenate of strychnia.*

Bryonia, with its symptoms of dryness in the mouth, high specific gravity of the urine, pruritus, loss of appetite and marked debility should be frequently studied in diabetes. *Creosote* has been recommended when tuberculous manifestations, with rapid emaciation, are in evidence, together with gastric and intestinal irritability. In younger subjects *ferrum phos.* and *calcarea phos.* may be called for in similar conditions. *Arsenic bromid, kali bromatum* and *asclepias vincet* have been highly recommended in the third or sixth dilutions. The last remedy was first studied in the veterinary schools of Vienna and attention was called to it by the fact that sheep, feeding on asclepias were subject to violent thirst and diuresis. Its relation to sugar elimination is not fully understood. *Plumbum metallicum* seems to have some relation to the disease when it is associated with albuminuria and intestinal colic with pronounced constipation. *Argentum metallicum* is claimed as an efficient remedy when diseases of the bones or specific degenerations precede the diabetes; it is useful when a profuse and turbid urine, with rheumatic complications and a persistent neuralgia are present. *Glonoin, jaborandi, helonias, lycopus, silicea, natrum mur.* and the *salts of lithium* have been suggested when their characteristic indications are present. The best remedy, which may be used homeopathically, is *syzigium jambolanum,* which is the active principle of jambul seeds. It may be given in the first decimal trituration or in ten grain doses of the powdered seeds or tincture. The maximum dose may be administered four to six times daily and gradually increased according to its endurance.

When there are signs of hepatic disorder, we may resort to remedies which have a decided action upon the liver. In this respect such remedies as *leptandrin, chelidonium, chionanthus, carduus, podophyllum, bryonia* and *nux vomica* may be considered.

DIABETES INSIPIDUS.

This is a disease in which polyuria and persistent thirst are present, but sugar and albumin are not found in the urine. It is a constitutional disease which is supposed to have its origin in a chronic nervous disorder.

Etiology.—Patients under fifty are more commonly afflicted than those of advanced life and it is claimed to be a congenital possibility. *Males* are supposed to be affected more than females, though there is no rigid rule in this respect. It is invariably associated with nervous influences, and injuries which affect the brain or spinal cord are predisposing factors. *Brain tumors*, or any lesion which irritates the region of the floor of the fourth ventricle, are liable to develop it. Paralysis of the sixth nerve, with or without meningitis, is also said to be a cause. *Tuberculous meningitis,* epilepsy and hereditary syphilis are looked upon as causes. The disease may also appear as a sequela of saccharine diabetes after sugar has disappeared from the urine. *Abdominal tumors* near the celiac plexus, incontinence of urine, and tapeworm are also regarded as causes. It exists as a sequence of acute infections and in this relation it occurs most frequently during the stage of febrile convalescence. Diphtheria, measles, scarlatina and malaria may frequently provoke it. *Intemperance* is regarded as a common incitant and the daily consumption of large quantities of beer or even cold water is many times its cause. *Heredity* is also a prominent factor, and we may often trace it back to a neurotic inheritance. Exposure to cold, sunstroke, emotional disturbances and many other factors are claimed as the primary causes of its development.

Pathology.—No characteristic lesion of the disease has been found but there is no doubt of its relation to some nervous perversion. It is probably a vasomotor neurosis of central or reflex origin. It is a symptomatic occurrence when cerebral tumors or other brain lesions excite the region of the fourth ventricle. The kidneys may be enlarged and distended with blood, while the urinary tubules and accessory passages are often dilated. It may appear in persons of apparent robust health without any discernible cause.

Symptoms.—The characteristic symptoms may be noticed suddenly as the result of fright, emotional excitement or traumatism, or they may develop gradually. The principal feature is the *passage of an enormous quantity of limpid urine* of low specific gravity (1.001 to 1.005). *Thirst* is another prominent symptom and this often becomes distressing. Attending this there is a marked *dryness of the skin* and the absence of perspiration even upon exertion. The general health is not decidedly perverted except in protracted cases, or when the nervous exhaustion is extreme. The appetite is rarely voracious. *Headache,* restlessness, irritability and insomnia are frequently troublesome, but these are manifestations of the nervous complications, and not expressive of the disease. The *frequency of urination* is disturbing in itself inasmuch as it is often a requirement both during the day and the night. Diarrhea is sometimes an annoying feature and this, with the dryness of the tongue and mouth, are indicative of an associate alimentary disturbance. The disease has a tendency toward chronicity, though many cases are of short duration, depending, of course, upon the cause and extent of nervous derangement.

The symptoms are naturally suggestive of saccharine diabetes, but sugar is not found in the urine and the depletion of strength and the loss of flesh are not so pronounced. The amount of urine voided may frequently exceed that of true diabetes, though this is not liable to exist in the average case. The thirst, which causes the patient to drink so much water, seems to compensate for the liquid loss. The constituents of the urine are all increased with the exception of uric acid. Albumin is rarely found, though inosit may sometimes be detected.

Diagnosis.—The diagnosis will be made when the nervous causes are recognized and the quantitative urinary examination reveals only the polyuria, the low specific gravity and the absence of sugar and albumin. The differentiation in the case of true diabetes will not be difficult. Hysterical polyuria will appear only during hysterical manifestations while the polyuria of chronic interstitial nephritis will reveal the presence of albumin, hyaline casts, together with the presence of arteriosclerosis and cardiac hypertrophy.

Prognosis.—The majority of cases are not of long duration and recovery may usually be expected. There are exceptions in cases of continued nervous predispositions and particularly when there is an organic development in the brain. Intercurrent diseases may cause unexpected death.

Treatment.—The first principle of treatment should comprehend the cause and, therefore, the nervous features will demand unusual attention. If the habits, vocation or environment tend to produce the neurasthenic state, correction in this respect should receive proper attention. The general treatment for neurasthenia will apply in full force. The thirst is caused by the diuresis and, therefore, it seems best not to curtail the ingestion of a normal amount of water, though cold drinks must be avoided. Over-drinking, however, becomes a habit and moderation should be applied as much as possible. *Hygienic measures* and healthful physical exercise are always to be recommended. The *diet* should also be regulated so as to encourage only a normal digestive function and at the same time furnish a proper amount of nourishment. When the excretion of urea is unduly increased nitrogenous food should be limited. *Alcoholic drinks* and coffee should be prohibited. *Galvanism* has had its advocates, and is serviceable in that class of cases in which spinal irritation is a decided factor. In such cases the positive pole is applied over the cervical vertebrae and the negative is placed over the lumbar region and the pit of the stomach alternately. *Tonics* and *nervines* should be used only when there is much physical depletion. Much care should be given to the removal of local irritations and for that reason it will be necessary to look for and relieve such possible conditions as masturbation, enuresis, tapeworm, phimosis, adherent clitoris and intestinal and rectal diseases. In many cases a spontaneous cure may take place without any local or general treatment.

Internal medication has not received special attention since the remedies must naturally apply to the symptoms of the provoking disease. *Ergot* was highly lauded for a time and it is presumed to have cured some cases. When employed the dosage has varied from 10 to 20 grains, three to six times daily. There are many objections to this drug and it is questionable if it will give permanent relief. *Opium* has also been lauded for the same reasons given under its use in saccharine diabetes, but it hardly applies in this disease except as it refers to the suppression of urine. *Gallic acid* has also been recommended, often in combination with opium, for its astringent effect. It seems to be useful, however, only when the irritation is induced by cystitis and an associate chronic diarrhea. Some value is attached to its potency effect. *Sodium bromid,* in gradually increasing doses, has been recommended, as has *potassium iodid, strontium iodid, jambul, antipyrin, cod liver oil,* all of which, with the possible exception of jambul, seem to have been used in a blind effort to give something without knowing why.

The proper treatment will consider the conditions and the individual symptoms of each case. The following remedies may be employed in accordance with their homeopathic indications: *Valerian, valerianate of ammonia, asafetida, ignatia,* and *crocus sativa,* are naturally prominent when neurasthenic symptoms are pronounced. *Strychnia phosphate, phosphorus, phosphoric acid* and *china* are of value when the nerve depletion is pronounced, and phosphaturia, oxaluria or seminal wasting exists. *Ferrum phos.* should never be overlooked in anemic cases. *Scilla, apocynum* and *helonias* are remedies which refer definitely to the polyuria. The first named, in the second decimal potency, will often correct the frequent passage of a large quantity of pale urine; the second pertains more particularly to dropsical conditions and the thick urine which is frequently voided. The last named of these three remedies refers to a profuse discharge of urine, which may be saccharine, and the dragging sensations which represent either vesical or uterine congestion. *Strophanthus* is a remedy from which Goodno has obtained good results, and *murex* is also recommended when the urination is frequent at night and is associated with disorders of the female generative organs. *Argentum nitricum, aurum chlorid, picric acid* and *zinc* may be employed when the nervous disorder partakes of a degenerative character.

ARTHRITIS DEFORMANS.

This disease has been previously, though erroneously, defined as rheumatoid arthritis and rheumatic gout. It is characterized as a chronic deforming disease of the joints in which progressive changes in the synovial membranes, cartilages and bones, together with osseous periarticular formations, produce decided deformity.

Etiology.—Though the incipient arthritis may resemble the development of rheumatism and gout, it probably has no direct relation with either of these diseases. In accordance with the views of J. K. Mitchell and Charcot, it is of neurotrophic origin and the cause may be traced to some disease of the spinal cord. The following reasons are given as favoring this theory. The fact that arthropathies may occur in the course of tabes dorsalis, its frequent appearance in association with profound nerve shock, the symmetry of the deformities of the joints, the muscular atrophy, the contractures, and the skin dystrophies manifested by pigmentation and local sweating. Falli also confirmed this theory by several autopsies in which he found atrophic changes in the anterior horns of the spinal cord, though he was not satisfied that the arthritis deformans was dependent entirely upon the nerve lesion. Traumatism has been considered as a cause; exposure to cold, and malnutrition from imperfect food, uterine disease and other causes have also been enlarged upon by various investigators. There may be some truth in the relation of uterine diseases inasmuch as women are most frequently afflicted. The microbic theory has had some advocates and Dor claims that the germ is "an attenuated culture of the staphylococcus pyogenes aureus."

The *predisposing* causes are quite numerous, though nervous shock, grief, mental worry and prolonged neurasthenia are the most common. Uterine disorders, as mentioned above, are frequent precursors. *Age* must be something of a factor, as it usually starts in the early part of life (between twenty and thirty). The disease has been discovered in advanced life and when it occurs in patients under twenty, it partakes of the acute form. *Heredity* is an influence, since the disease may be shown to be a family tendency. The debilitated furnish the most cases and, therefore, it is without doubt a feature of poverty. *Infectious diseases* are regarded as favorable influences inasmuch as it may be so frequently discovered subsequent to their occurrence.

Pathology.—The pathological changes begin in the cartilages. These, following cell proliferation, fibrillation and softening of the matrix, undergo a process of absorption in the center, which permits the bones to come into contact at this point. As the bone ends are made bare, they may undergo atrophy from the constant attrition and become smooth and eburnated. At the edges of the cartilages, where the pressure is less pronounced, a gradual thickening takes place, with a subsequent ossification and the formation of osteophytes which, overlapping the articular surfaces, tend to lock the joints. Bony outgrowths from the periosteum of the shafts will favor the joint defect. The synovial membranes, becoming inflamed, also take on a cell proliferation with organization and sometimes ossification of the exudate. The capsule and ligaments become thickened later and aid in producing the pseudo-ankylosis. The abnormal structures may undergo softening and permit a partial or complete luxation. Fragments of the cartilage are often detached and remain loose in the joint. Muscular atrophy may also be a feature of the disease.

Symptoms.—There is a rare form of the disease which presents *acute symptoms.* This usually appears in children or young people and is characterized by a multiple arthritis which affects both the large and small joints simultaneously. *Pain* is marked, there is slight redness of the parts involved and considerable swelling exists as the result of an effusion within the joint. The *febrile disturbance* is a characteristic feature. The increase in the size of the joints may be associated with progressive enlargement of the lymphatic glands in the neighborhood of the joint and also enlargement of the spleen.

The symptoms of the *chronic form* are most expressive of the disease. Swelling starts at first in one or more finger-joints and very soon corresponding joints of the other hand are affected. *Pain* exists in case of movement and, as the enlargement gradually increases, the joints become immovable. There is *slight redness* and *tenderness* and, during the early stages, the symptoms may show marked exacerbations with occasional tendencies toward improvement. Gradually a similar involvement of the feet, arms, legs and trunk will be detected. The fingers are usually deflected toward the ulnar side, and more rarely toward the radial side. They are also partially flexed and sometimes overlap each other. Luxation is an occasional development, but the joints are more liable to become fixed, though a limited degree of motion may exist for some time. The thumb is rarely affected. Palpation may detect *crepitation* when the joints are forcibly moved. The adjacent muscles undergo *gradual atrophy* and, as they become the seat of contractures, the limb may become flexed which adds to the deformity and impaired function. *Trophic changes* are pronounced and pigmentation of the skin, together with other neurotic disturbances, are frequently observed. The pulse is usually rapid and perspiration may be troublesome. The morbid changes may be confined to a few joints and after a time the progress of the disease may be apparently arrested. When it assumes the progressive form *constitutional symptoms* are liable to appear and in such instances the signs of gastro-intestinal disturbances and secondary anemia are in evidence. Nervous symptoms may develop into hysteria or pronounced hypochondriasis.

Clinical Varieties.—Chronic arthritis deformans may be found in several forms. The *multiple form,* also known as *Heberden's nodosities,* is characterized by nodules which develop on the sides and ends of the distal phalanges of the fingers and sometimes on the toes. In this form there is no apparent tendency for other joints to be involved and it is not a formidable disease. Women in middle life are frequently predisposed and it is erroneously confused with the arthritic accretions of gout. Pain and tenderness may be experienced when the hands are cold, but the deformities are not generally the seat of much tenderness. A *progressive form* with acute and chronic manifestations has also been recognized. In the *acute* development it simulates a mild form of rheumatic fever; the joints are swollen and tender, while fever characterizes the initial stage. Periods of improvement alternate with exacerbations of the early symptoms as the disease progresses toward permanent enlargement and deformity of the joint. The *chronic* form is recognized by a slower development and the absence of fever and other acute signs. The symmetrical enlargement affects the hands, knees, feet, ankles, wrists, elbows, shoulders, jaws, hips and sometimes the cervical and dorsal vertebra. The knees are often so enlarged and fixed that the legs are flexed upon the thighs and the thighs upon the trunk. Contractures also both causatively precede these flexures and develop with, or after them. The wrist may be so deflected and the elbow may take such a rectangular bend that marked deformity exists. The wasting of adjacent muscles is inclined to exaggerate the appearance of the joint enlargement. Effusion may also take place into the joint. Numbness, tingling, skin pigmentation, local sweating and pain attend the gradual development of the joint ankylosis.

The *monarthritic* or partial form is observed most frequently in men of advanced years and it often succeeds an injury. The hip joint is frequently involved, when it is known as *morbus coxae senilis.* The other joints affected are the knee, shoulder and the vertebral articulations. The pathologic and clinical features are similar to those of the polyarticular variety.

Diagnosis.—The pathological findings are absolutely essential for the making of a correct diagnosis. It is distinguished from *gout* by the absence of tophaceous deposits. *Acute articular rheumatism* is differentiated by the fever, the red, swollen and painful joints, the marked sweating, the cardiac complications and other distinctive etiological and pathological characteristics. In the chronic features of articular rheumatism diagnosis is more difficult yet in this only a few of the large joints are involved, the deformity is not the same, ankylosis is not so marked and a symmetrical involvement is not observed.

Prognosis.—A cure by treatment is not to be expected in advanced cases, though it is possible to limit its development if the underlying cause is found, and early treatment is instituted. Associate complications, particularly anemia, may be overcome by remedies and the severity of the symptoms may invariably be alleviated. Spontaneous arrest of the disease is possible and when this occurs, the prognosis so far as life is concerned, is always favorable.

Treatment.—While it may not be expected that treatment will have much influence upon the pathology of advanced cases, it is evidently true that we may accomplish much by the early symptomatic treatment with adjuvant hygienic care. The nutrition must be improved by means of highly nourishing food and to this end the diet, general habits, occupation and environment of the patient must be regulated. Most physicians advise a journey to some of the springs and undoubtedly this is of value in many cases. A change of climate is always invigorating. Some authorities emphasize the value of foods which are rich in fats and claim good results therefrom. Galvanism has been advised and probably has some influence over the pathological changes but sufficient data in this respect are not at hand to warrant very definite conclusions. Hot baths, particularly the Turkish bath, and the employment of superheated dry air may be beneficial in many cases but we must not expect too much from these measures. Fibrous ankylosis may demand surgical measures. Massage and osteopathic manipulation may be of service also. When pain is pronounced, hot compresses covered with oiled silk may give great comfort.

The internal medication is to be given with regard to the symptomatology. Goodno is inclined to the use of *colchicin* for the pain in the finger-joints and the subacute attacks which affect other joints. He gives a solution of Merck's colchicin—1 grain to the ounce of alcohol—three drops every two or three hours. The value of this remedy seems to apply to the rheumatic conditions rather than to the typical arthritis deformans. *Benzoin, benzoic acid,* the *benzoates of lithium* and *ammonium* are also highly recommended. *Arsenic, calcarea phos., ferrum phos.* and remedies of this character should be thought of when anemia is a distinct factor. *Pulsatilla,* in ten drop doses of the tincture, may be given every two or three hours with much relief when there are swellings of the larger joints. *Belladonna, rhus tox, hamamelis, bryonia* and *cedron* may be compared, and the majority of these may be used in the low potencies alone. *Sepia* has been recommended when there is portal or uterine congestion, and it would seem also to apply to the pathological changes in the lower extremities, with pains extending up the back. *Collinsonia* will be indicated when there is constipation or hemorrhoids, and there is a tendency to enlargement of the smaller joints. *Sulphur, esculus, aloes* and *hamamelis* may be compared with this. *Cimicifuga* covers the myalgic symptoms which are usually defined as rheumatic; it pertains also to the twitchings and jerkings of the muscles and to an underlying neurasthenia. *Gelsemium, agaricin, cuprum* and *camphor* may be compared.

Such remedies as *iodid of iron, iodid of sulphur, chlorid of gold* and *calcarea sulph.* are no doubt of value when their indications are clearly established. *Ammonium phos.* has as one of its chief symptoms "nodosities of the joints." *Causticum* is also a remedy which has an affinity for arthritic affections, though usually of the rheumatic character. *Guaiacum, mezereum, ledum, mercurius, ruta, manganum* and *rhododendron* are remedies worthy of careful study in each case. Any remedy which tends to restore the general nutrition is always useful. In my own experience I have been satisfied that the best results are obtained when the disease is treated from a neuropathic standpoint. In a somewhat extended experience in my clinic during the past ten years, I am convinced that this is the best procedure. Those cases which I have treated in accordance with the nervous theory of the disease have certainly shown marked improvement when they persisted in the treatment with any degree of regularity. I am quite confident that our success in the care of incipient cases of this disease will depend upon the early and persistent treatment along this line. *Picric acid,* in the third decimal potency, four to six times daily, has given me better results than any other remedy. The failures, I believe, are due to the fact that it is not given long enough. *Thiosinamin* should be thought of and given a trial in this disease.

GOUT.

This disease is unfortunately too frequently confounded with rheumatism. It is an acute and chronic constitutional affection due to a perverted nutrition—possibly the result of autointoxication—following which there is an abnormal accumulation of uric acid in the blood and tissues with a resultant arthritis which is the most distinctive feature. Disordered metabolism due to the lack of physical exercise and defective elimination of waste products are contributing factors in the development of the disease.

Many theories relative to the uric acid accumulation have been advanced to account for the disease, but it is still a difficult task to settle the question whether it is an increased formation or a deficient elimination of this product which causes the arthritic trouble. The toxic cause is probably traced to the allied products of uric acid. Garrod claims that the acute attacks are produced by an excess of uric acid in the blood, a decreased elimination and a deposition of urate of sodium in the joints. Haig holds to the theory that it is not due to an excessive formation, but rather to the retention of uric acid in the blood while its deposition is due to the fact that there is a reduced alkalinity of the blood. Pfeiffer reasons that both gout and gravel are due to the production and excretion of uric acid in a form which is difficult of solution. Sir William Roberts believes "that acute attacks of gout are dependent upon the precipitation of the crystalline biurate of sodium and that the urate is transformed into the less soluble biurate in the blood." Ebstein ascribes the excess of uric acid in the blood to an abnormal formation of this substance in such parts as the bone marrow and cartilages which do not normally produce it. The lymph and the blood thus become surcharged and relief is only obtained by decomposition of uric acid and through excessive renal activity. When the lymph moves slowly the premonitory symptoms of gout appear and when complete stasis exists the acute attacks take place. The concentrated solution of uric acid then acts like a chemical poison on certain tissues and is followed by necrosis with a deposition of sodium biurate. If this stage is not reached local arthritic symptoms are not pronounced. Sir Dyce Duckworth advances the nervous theory, claiming that the diseased state is dependent upon a primary neurosis. Many other theories exist, but all are related to the fact that uric acid is the factor for consideration both in the systemic manifestations and the local arthritic disturbance.

Etiology.—*Heredity* may be blamed for many cases and without doubt the transmission of the disease may be frequently traced back several generations. *Age* has something to do with its occurrence for the primary attacks are rarely observed before puberty and the majority of cases develop during middle life. In exceptional cases it may occur in very young children, and those of advanced years are occasionally susceptible, but as a usual thing it decreases in frequency after the fiftieth year. *Sex* is also a factor inasmuch as it develops most frequently in men. Women suffer less from the arthritic form, but they are liable to attacks of irregular gout, particularly at the time of puberty or the climacteric. *Diet* must always be regarded as a causative influence, since over-eating and indolent habits—the crowning faults of luxury—particularly when associated with deficient physical exercise, are almost invariably discovered in the history of each case. Nitrogenous food is most at fault, but even a mixed diet, when the indulgence is intemperate, may lead to the same result. Gout is usually thought of as a visitation to the glutton and the analogy is no doubt true. *Alcohol* is another influence which favors the disease and its companionship with gluttonous habits without doubt incites the acute attacks. The disease is most prevalent in communities where the heavier malt liquors are indulged in. *Occupation* and *social position* are also named as inducing causes and we shall always find it prevailing not alone in the aristocratic class, but as well among those whose vocations and associations encourage over-eating and an insufficiency of physical exercise. The social position, therefore, is always an element in the etiology of the disease, yet we must always keep it in mind that poor man's gout is a possibility. *Lead poisoning* is given much consideration in relation to this disease and many investigators have freely maintained that those who are afflicted with this are subject to gout. These causes already enumerated do not alone constitute the etiology of this disease, for we shall find that pathologic conditions which affect other organs—particularly the liver and the kidneys—may lead to a disturbance in the excretory processes which favors this metabolic change.

Pathology.—The study of the morbid anatomy does not give an opportunity for much else than the consideration of the arthritic changes. The *deposition of uric acid* in the cartilages, ligaments and synovial membranes of the joints occurs at first in the fluid state with the appearance also of minute crystalline masses. As this exudate becomes hard and dry, the formations are recognized as *tophi.* Secondary inflammatory changes lead to fibrous overgrowths and eventual distortion and fixation of the joints. *Partial absorption* may occasionally diminish the joint involvement, though the ulcerative process is more liable to complicate the local condition. The *digital joints* suffer most from the early invasions, though the depositions may invade other joints and tissues, a chalky formation being found even in the cartilages of the ears, nose, eyelids and larynx; the bones underneath the cartilages may be occasionally the seat of the deposits. The fingers are often deflected as the disease progresses and similar symptoms are observed in the toes. The most distinctive feature of the acute attack—in true podagra—is the painful inflammation of the great toe.

The kidneys usually display a similar pathology and areas of necrosis, with secondary uratic deposits, are observed in the renal parenchyma; these are found most frequently in the papillae, though the degenerative changes are liable to invade a greater portion of the renal structure. The granular contracted kidney is an occasional sequence of the disease. The *heart and blood vessels* undergo pronounced changes. The deposition of uric acid may affect the valves of the heart, but cardiac hypertrophy is the most common pathologic condition following an arteriosclerosis which is induced by the faulty elimination in gout. Emphysema, asthma and chronic bronchitis are rare complications found in the respiratory tract.

Symptoms.—*Acute gout* will be recognized by the appearance of arthritic symptoms. These are often preceded by prodromal manifestations which presage its development. Disturbances of the gastro-intestinal tract, mental and nervous irritability, muscular cramps and articular pains may be suggestive of the later appearance of paroxysmal and excruciating pains which characterize the true joint involvement. The amount of uric and phosphoric acid elimination in the urine may be diminished at this time, though these are not regarded as typical or uniform features. *The attack* is characterized by severe articular pain most pronounced in the great toe at the metacarpophalangeal joint. The seizures often occur at night or in the early morning and sometimes may be ushered in by a chill. *Heat, redness, swelling* and excessive sensitiveness are soon experienced, while the skin becomes shiny and often pits upon pressure. *The temperature* may rapidly rise to 102° or 103° F. *Acute pharyngitis* or "gouty sore throat" is occasionally an early complication. These symptoms may suffer a remission, only to be followed by excruciating suffering after a brief interval. The repetition in these attacks usually ends in some moderation in the paroxysms until desquamation of the skin takes place and the general health assumes its previous condition until there is another recurrence.

There may be a sudden *transmission* of the arthritic process to some internal organ. In such instances there will often be experienced a quick termination of the joint symptoms with an equally intense pain in the region of the stomach; vomiting, diarrhea, faintness and circulatory disturbance will then be evident. The *heart* may likewise be suddenly affected and precordial pain, dyspnea and palpitation will cause great suffering. Should the *brain* be similarly affected, grave cerebral symptoms with other nervous phenomena may result from the uremic infection. *Cystitis,* prostatitis, orchitis or parotitis may occur as the result of retrocedent gout. The urine is scanty, acid, highly colored and of high specific gravity. Uric acid and urates are deposited when it is allowed to stand and a small quantity of albumin may be present.

Chronic gout appears after the gradual subsidence of the acute symptoms. It will be characterized by the relief of the local inflammation and the implication of corresponding and remote joints; the fingers, toes, wrists and ankles may be extremely affected, though the tophaceous deposits rarely invade the larger joints like the hips and shoulders. As the chalky deposits slowly increase, the deformities develop until locomotion and the use of the hands are limited. The associate conditions of arteriosclerosis, gastric disorders, cardiac hypertrophy and contracted kidney gradually develop. The urine in this stage is liable to reveal the presence of albumin and tube casts, while a recurrence of the acute exacerbations, with all their dangerous complications, may suddenly take place at any time.

Irregular or atypical gout is characterized by a distinct set of symptoms which are suggestive of gout or a gouty diathesis and an hereditary tendency in this respect is usually discovered. Evidence, however, of an arthritic attack is not generally found, though it is possible for acute gout to succeed or be associated with this condition. The features of irregular gout are recognized by many peculiar symptoms: *Joint and muscle pains,* particularly in the back and thighs, are typical of this condition. The patient complains of distressing wandering pains in these regions, and it is not uncommon for these to be located in or near the joints, which, however, are not enlarged.

Gastro-intestinal disorders may be recognized by symptoms identical with those of lithemia. Arteriosclerosis likewise may exist from this cause as the uric acid in the blood raises the arterial tension. *Nervous manifestations* will also be recognized by the occurrence of migraine, sciatica and neuralgia which are expressions of a gouty neuritis. Such forms of paresthesia as tingling, itching and burning sensations may occur with or without the cutaneous eruptions which resemble urticaria or eczema. *The urinary symptoms* which refer to irregular gout will be defined by the highly-colored, scanty urine of high specific gravity, together with the so-called "uric acid showers" in which uric acid is increased perhaps to be followed by deficiency in that respect. Gravel, intermittent glycosuria, oxaluria and interstitial nephritis may develop as a sequence of this condition. *Pulmonary disturbances* like bronchitis, asthma and emphysema are occasionally found and *ocular disorders* like conjunctivitis, keratitis, iritis, hemorrhagic retinitis and glaucoma are not uncommon results.

Diagnosis.—Typical gout does not present any difficulty in diagnosis, though it will require careful investigation to determine the irregular form. The differentiations between this and *acute rheumatic fever* is also comparatively easy unless the gouty affliction takes the form of polyarthritis. The history and the development are different and acute rheumatic fever will be distinguished by its characteristic fever and the painful swelling of the larger joints. *Chronic rheumatism* will be differentiated by the fact that it affects larger joints and has none of the secondary conditions like interstitial nephritis and arteriosclerosis. *Arthritis deformans* involves the fingers more than the toes, develops symmetrically, pursues a progressive course, has no acute attacks, the deformity follows an exostosis and ankylosis, uric acid is not a factor and there are no associate complications.

Treatment.—The treatment involves first of all a painstaking regulation of the diet and habits. The majority of these patients are afflicted with too good an appetite which they augment too frequently by the use of stimulating drinks. Other habits may be equally encouraging to this disease and their correction will be necessary to put the patient into a position to begin a proper treatment. Regular meals are almost as essential as the regulation of the diet. Plenty of sleep, sufficient outdoor exercise, a relief from an exacting business or domestic strain, a careful application of hygienic principles and every influence which favors a simple life must be insisted upon before the direct treatment is instituted.

The *dietetic regulation* will require that the patient be confined to a form of food which produces a minimum of uric acid. This will mean the reduction in amount of all nitrogenous foods which, by their complete combustion, furnish urea and by their incomplete combustion furnish uric acid. While it is not always practicable to completely eliminate these foods, they may at least be administered cautiously. This will exclude beef, veal, mutton, lamb, pork, and, with some exceptions, fish; oysters and lobsters may be used sparingly while the white meat of the fowl may be used quite freely. It is generally accepted now that the nuclein foods should be prohibited. Fats are a good form of food, milk is the best diet and stale bread will be digested easily. The diet which is safe, therefore, is reduced to milk, butter, fruits and the succulent vegetables, with various modifications and additions as may be tolerated from time to time. Some are inclined to interdict the use of carbohydrates, but while their restriction may be required, it may be safe to permit their use, providing proper limitations as to over-eating are demanded of the patient.

The *most exacting limitations* must be applied to such stimulants as malt liquors and wines. It is not alone the alcoholic effect which these produce, but the unnatural stimulation of the appetite invariably causes gluttonous habits. Therefore, there should be no tolerance of any of these beverages. While perchance some liquors may be used with slight evil results, unless the habit is completely broken, the patient will have periodic seasons of backsliding when all the good work obtained by an abstemious interval will be undone. Very acid fruits cannot be tolerated by some gouty patients, while others suffer no bad results from them.

Natural *mineral waters,* next to diet, are the most essential features of treatment. Ordinary distilled water will usually suffice, but it does not make a sufficient mental impression upon the patients to compel them to use it freely. The best results are obtained when a spring is visited, particularly if it is popular and the surroundings are encouraging to a careful diet, a quiet life and a sufficient use of pure water. The alkaline waters are most favorable to a cure in the majority of cases, but some may need a combination of the aperient principle as well. The saline waters are also useful in many cases and we have a variety of such springs, both in Europe and in this country. The salts of many of these springs may be successfully employed when it is impossible for the patient to leave home. Under the *hygienic treatment,* we must not forget the adjuvant help of the bath. Elimination is doubtless encouraged by the regular hot bath, but a more perfect skin function will be obtained by the morning plunge or cold or tepid shower. Of equal importance with this is an outdoor life and a moderate amount of healthful physical exercise.

The *treatment* of the *acute arthritic attack* is the first requirement made upon the physician, for the patient rarely realizes any trouble until this appears. In order to give this the proper attention our patient should be sent to bed and compelled to remain there until all signs of the acute inflammation have subsided. This precaution will surely shorten the attack.

By reducing the diet absolutely to liquids, the use of plenty of alkaline drinks and the internal administration of the indicated remedy, it is possible to ameliorate the usual paroxysm until a fixed plan of diet and treatment can be entered upon. Such remedies as *aconite, bryonia, belladonna, ranunculus, rhododendron, rhus tox, colocynth* and *gelsemium* will generally be demanded. For the acute pain and swelling *colchicum* has been a standard remedy for some time. It has been used in potency or in the tincture (5-20 drops) every two or four hours. In many cases it will alleviate the pain after a few doses have been given and then it is advisable to use it less frequently and in smaller doses. There are many objections to its administration in such large doses and many cases will not tolerate it. Therefore, it is wise under all circumstances to consider the lower potencies. The quick action of the tincture is due, no doubt, to its prompt stimulation of the hepatic secretion. Thus it may be proper in many cases to give the tincture for a few doses in the beginning and then continue the remedy for some time in potency. It was an old theory that colchicum checked the formation of uric acid, but this was probably surmised inasmuch as it is a stimulation to excretion and thus it promotes the elimination of other waste products as well as uric acid. Objections have been offered on account of its being a vascular depressant, but there is no need of this occurrence if proper watchfulness for cardiac weakness is maintained, as it should be, under all circumstances. *Colchicin,* in the third decimal potency, may be a more serviceable form of the remedy. The *salicylates* have been recommended by many experienced practicians, but their chief indications will be found only in rheumatic subjects. It must be remembered, however, that their action as hepatic stimulants make them useful for the elimination we are seeking. Again we are well aware that in arthritic pain they are certainly indicated. The *salicylate of sodium* is probably the best form to employ and it should be used in doses large enough for its drug effect. Fifteen grains four times daily, for adults, is none too much, though in some cases it may be better to use ten grains every two hours. As soon as relief is obtained, the dosage should be gradually reduced. The *alkaline carbonates,* in divided doses, should supplement the salicylates. The *citrate of potash* and many of the milder salines may be used until there is free bowel action.

There are many so-called *uric acid solvents* on the market, some of which are good and some are not. All of them obtain their repute by their action upon the liver. I have used a mixture containing iris versicolor, apocynum, citrate of lithia and the acetate of soda with decidedly good results. Its chief action is upon the liver and that I attribute mostly to iris, which is my favorite hepatic remedy. Therefore, it is right for us always to take into consideration, for acute attacks, such remedies as *iris versicolor, chelidonium, chionanthus, carduus* and *mercurius dulcis.*

Several other remedies have been advised for the acute attack. *Piperazin* is supposed to be a solvent of uric acid, but its efficiency depends probably upon its diuretic stimulation. It is administered (grs. 5-10) two or three times daily, freely diluted with water. The *lithium compounds*—the carbonate and citrate—have been employed quite extensively, but their utility is problematical. Five grain doses—three times daily—with an abundance of water, is the best method of administration; the potassium salts may often be added. *Guaiacum* has been used with some degree of success, but its best results are obtained in chronic conditions of gout, and the same may be said of *potassium iodid.* The hypodermatic use of *morphin,* though highly commended by some, should be used only as a last resort to relieve pain.

Other remedies may be given according to their symptomatic indications. *Berberis* is often indicated when there are signs of oxaluria, or catarrhal attacks of the genito-urinary tract. *Benzoic acid* and the *benzoate of ammonium* may be considered in the same relation when the urine is deficient, high-colored and strong in odor. *Pulsatilla* may be efficient when the arthritic pain has a tendency to shift rapidly and it also applies in females with uterine disorders and when there are pronounced digestive disturbances. With this we may always keep in mind *cimicifuga, senecio, aletris* and *helonias.* No remedy refers better to the acute gouty attack than *ledum*; it is indicated when the gouty pains due to swelling are located in the small joints. This remedy has also a close relationship with *ruta graveolens, hamamelis, bellis perrennis, arnica* and *kalmia.* When gastric or intestinal colic are symptomatic features we shall often get relief by the use of *plumbum metallicum* or such associate remedies as *alumina, platina, opium* or *thallium.*

Gnaphalium is another valuable remedy, the chief indicating symptom, however, being an intense sciatic pain which alternates with numbness. It is also applicable in the gouty pains in which both sciatica and colic are also factors. It is properly compared with *ammonium benzoate* and *staphisagria.* Many other remedies present a symtomatology which is worthy of careful consideration. For instance, there are many cases which present the picture of the febrile action and gouty soreness which are suggestive of *eupatorium perfoliatum;* or the periodic, lancinating joint pains of *cedron* or *china*; or the hot, swollen joints of *bryonia;* or the herpetic pains of *sepia.* And so we may enumerate many other remedies which portray a usefulness through the individuality of their symptomatology.

The *local treatment* of the arthritic inflammation may be advisable. The parts should be wrapped in cotton or flannel and put at rest, the same as in rheumatic fever. Hot alkaline solutions, or possibly hot fomentations, may be serviceable. A lotion containing half an ounce of carbonate of soda, and two drams of laudanum to ten ounces of water, may be prescribed. A portion of this should be diluted again with an equal part of hot water and applied to the parts on saturated pieces of lint and covered with oiled silk. In many cases complete relief may be obtained in the same way with wet dressings of a saturated boracic acid solution.

LITHEMIA.

Lithemia is a disease closely related to gout. The condition is no doubt the result of faulty oxidation of nitrogenous matter and is characterized by an excess of uric acid in the blood, together with certain digestive, circulatory and nervous phenomena. It differs from gout inasmuch as there is an absence of joint deposits and joint inflammation. It has also been defined as uricacidemia, uricemia and American gout, the last term being employed because the disease is presumed to take the place of gout as it exists in England. Impaired elimination is the characteristic feature of the disease and this will exist when intemperate eating or drinking are associated with a lack of physical exercise allowing uric acid to be stored up in various tissues of the body. In other words, the "income of nutriment is in excess of the output of waste," and as a result the accumulation gives rise to the morbid phenomena of the disease. Males are mostly predisposed, while alcoholism and heredity are contributing etiological factors. Climate has also an influence inasmuch as the colder climate favors diminished skin excretion.

Symptoms.—The symptoms are somewhat similar to those of irregular gout. The *gastro-intestinal conditions* are characterized by indigestion with fickle appetite, which at times may be voracious and again impaired to the extent that no food is desired. Gastric discomfort with a sense of fulness after meals is a frequent complaint, while nausea, vomiting, extreme gastric acidity, and an unpleasant metallic taste in the mouth are frequent disagreeable features. The *bowels* do not act regularly and the bile is often absent in the feces; flatulence and intestinal discomfort are persistent disturbances and in many cases diarrhea alternates with constipation. *Aphthous ulcers* may be found in the mouth and on the tongue, while hemorrhoids evidence the hepatic disorder. *Neurasthenic symptoms* are quite pronounced at times while vertigo, with headache, is frequently distressing. *Cardiac palpitation* may be experienced after eating, but otherwise the pulse is characteristically slow, with increased tension and at times there is marked accentuation of the aortic second sound. *Paresthesia,* together with the sudden appearance of muscular pains in various parts of the body, is a common complaint. In addition to the above we shall find that the *depression of spirits,* with a constantly increasing tendency toward melancholia, is a most trying condition to treat. Variations in the amount, color and specific gravity of the urine are common and a deposit of mixed urates and uric acid are frequently observed when it is allowed to stand; this, however, is by no means a distinctive feature in every case.

Diagnosis and Prognosis.—To make a positive *diagnosis* it will be necessary to take into consideration all of the etiological features and the symptoms which define the difference between this disease and gout. Heredity, occupation, habits of overeating and drinking, together with the condition of the urine, will usually emphasize the defective oxidation. It will be confused most frequently with gout, and this differentiation will be determined by the absence of arthritic symptoms; it is possible, however, for the two diseases to coexist, or at least to be so closely related that a distinction can not be easily made. The *prognosis* will depend upon the patient, the duration of the disease and our ability to overcome the imperfect oxidation by diet, improved habits and remedies. Compared to gout, it is usually more responsive to treatment.

Treatment.—The treatment of lithemia does not differ essentially from that prescribed for gout. *Intemperance* in eating and drinking must first be regulated. Meats should be eliminated from the diet as much as possible and not cause weakness. Fresh green vegetables will be tolerated, though in case of much fermentation it may be necessary, for a time, to employ a limited nitrogenous diet. Oysters, fish, eggs and the white meat of fowls may usually be allowed. Sugar must be restricted and fats are not generally well borne.

Exercise is an essential feature of the treatment. If the vocation is such that the patient has no incentive to physical exercise and particularly if there is a tendency toward a sedentary life, it will be necessary to prescribe and insist upon something of this kind. Athletic exercise is easily found in horseback riding, golf, tennis, rowing and walking. In the winter it will not be so easy to find equivalent exercise, but skating, massage, osteopathic treatments and the popular forms of physical culture may be substituted. *Water* is the next adjuvant of importance. The patient should be encouraged to drink freely of the lithia waters which are on the market. When these can not be afforded, pure spring water may be substituted. In case of *constipation* the saline or aperient waters may be employed, as catharsis—if not carried too far—is a necessary feature of treatment. The Saratoga waters, Hunyadi, Friedrichshalle, Rubinat, Pluto and many similar waters may be used successfully. Sodium phosphate, Carlsbad, Lithium and Thialian salts are often serviceable, inasmuch as their condensed form is a convenience. The effervescing lithia tablets may be used when the above are not to be obtained. In arthritic complications it may be necessary to employ the salicylates and other indicated remedies.

The *internal remedies* will be demanded in accordance with the symptoms. When hyperchlorhydria is a factor it will be necessary to use an *alkali* to correct the gastric perversion or when the acidity is due to organic acids *hydrochloric acid* will give much relief. *Nux vomica, pulsatilla, china* and *arsenicum* will be indicated when tonics are demanded. *Lycopo-*

dium, carbo veg., plumbum aceticum, moschus, sulphur and *sepia* will often be serviceable when gaseous distention is present. The nervous phases will be met by *conium, nitric acid, aurum, sumbul, asafetida,* the *zinc* salts and similar remedies. When the liver needs attention *chinoidin, carduus, iridin, thlaspi bursa pastoris* and other hepatic remedies will be required. Aside from the dietetic treatment and the other adjuvant recommendations it will be best to study carefully the individual suggestions in each case. The general care of the case does not differ essentially from that required in gout.

RACHITIS.

Rachitis, or rickets, is a constitutional disease of childhood characterized by impairment of the general health, arrested physical development and deformity of the bones due to cartilaginous overgrowth and delayed calcification.

Etiology.—The exact cause of rickets is not known, though it may be invariably traced to definite conditions. *Environment* is the most prominent influence, as we usually find rachitis among children in the poorer quarters of our large cities where filth is supreme and sunshine rarely appears. It is an exception to find a case among the more fortunate class of people unless there is a specific cause, a faulty heredity or acquired conditions which predispose to malnutrition. It may occur in the newborn, though it is inclined rather to a gradual development in the early period of child life. *Syphilis,* though not a direct factor, may be a suspected cause of its congenital appearance. While rickets may not be inherited as a disease it is liable to exist when the parents suffer from physical depravity. It may have its origin in defective breast milk, particularly when it is deficient in phosphates and fats, and the prolonged nursing by an overworked and underfed mother is a potent factor. It is also of common occurrence among bottle-fed children when the cow's milk is poor in fats or the prepared food does not meet the demands of nutrition. The lack of proper sterilization and the uncleanliness which generally attends in such instances, often incite its development. A *deficiency* in *proteids* and fats when the child has passed the nursing age and possibly the excess of farinaceous foods may encourage its development. At any rate the disease is always fostered when the food is insufficient or does not contain the proper principles for the nourishment and development of the growing child. The geographical distribution shows a larger percentage of cases in Europe than in this country, while it occurs more frequently among the colored than the white race.

Pathology.—The *characteristic lesions* are found in the bones, particularly the ends of the long bones, the ribs and the cranial bones. According to Osler a slight bulging is observed between the shaft and epiphysis and upon section the zone of proliferation is greatly thickened, bluish in color, irregular in outline and much softer than in the normal condition. The line of ossification is also irregular, spongy and decidedly vascular. The periosteum is easily stripped off and underneath this will be found a peculiar spongy tissue. As the result of these perversions of structure, ossification is delayed and imperfectly developed. The cranial bones also present areas of delayed ossification, particularly in the parietooccipital region—forming the so-called craniotabes, so that the bone yields to the pressure of the finger. This may cause depressions or flat hyperostoses may develop from the outer table, particularly in the frontal and parietal bones; although there may be a later improvement in ossification, these deformities may give rise to the broad forehead and prominent frontal eminences which simulate hydrocephalus. The change in the chondral ends of the ribs and the shape of the chest may also be pronounced. As the soft costal ends of the cartilages bend, the ribs fall inward while the sternum and cartilages are pushed forward; the lower ribs may be widened and the abdomen thrown forward by the arching of the vertebrae forward, thus giving the characteristic "chicken breast" or "keel-shaped" thorax. The clavicle may have an exaggeration of its normal double curve, the humerus may bend at the insertion of the deltoid, the radius and ulna may be twisted, while the long bones of the legs may be so distorted as to produce the "bow-leg" or "knock-knee" appearance.

The spine is liable to be curved outward in the cervicothoracic region or inward at the lumbosacral area, while a lateral curvature is also possible. The anteroposterior diameter of the pelvis is often lessened by the twisted and changed condition of the pelvis. Kassowitz considers the hyperemia of the parts involved to be the primary lesion which perverts the normal growth of the bone; the proliferation of the cartilage cells and the softening of the matrix thwart the development of normal osseous tissue. The newly-formed bone is always deficient in lime salts. The liver and spleen are usually enlarged and less commonly the mesenteric glands.

Symptoms.—The symptoms are not always suggestive at the onset. *Restlessness* and irritability are generally recognized early and the child resists being handled inasmuch as this causes pain. *Digestive perversions* and impaired nutrition are evident in the first stages of the disease. Convulsions frequently call attention to the fact that something is the matter with the child and in the severer cases we may not be surprised at the appearance of tetany or laryngismus stridulus. *Profuse sweating,* with a slight rise of temperature, is a characteristic feature and this alone, if a persistent symptom, is indicative of the nutritional disturbance which is typical of rachitis. *Dentition* is often delayed and when the teeth do appear they are usually subject to early decay.

As the disease progresses the *impairment of bone tissue* is identified by the peculiar deformities which may be detected. The cranium is apparently enlarged, since the bones of the face are greatly reduced in size. The sutures and fontanelles are not properly closed, and the head assumes a square outline with the anteroposterior diameter increased; this, with the pale and thin skin, gives the child an aged appearance. The ribs are beaded at the juncture of the ribs and cartilages—the *rachitic rosary*—the spinal curvature and the consequent changes in the thoracic outline tend to lessen the transverse chest diameter and thus interfere with the normal lung expansion. This explains the frequency of chest complications. The *characteristic chicken-breast* appearance, the prominent abdomen and the bowed legs are always present in well marked cases. The lower portion of the radius often suffers marked enlargement, the clavicle may present abnormal curving, the pelvis is frequently distorted, while the softening of the vertebrae and intervening cartilages account for the anteroposterior or lateral curvature of the spine.

Coincident with the typical skeletal changes we should give due prominence to the *increasing muscular weakness* and the attending pseudoparesis which has often been mistaken for genuine paralysis. Aside from the characteristic nervous features which may be associated with the disease, we may find many peculiar nervous phenomena which are a cause of much anxiety.

Diagnosis and Prognosis.—The symptoms already mentioned are sufficiently typical to fix the *diagnosis*. The appearance of craniotabes, sweating of the head, delayed dentition and other evidence of nutritional defects should call attention to the early phases of the disease. Tenderness in the lower extremities, with an indisposition to use these parts, is a very prominent symptom. Tenderness and enlargement of the wrists and ankles are also not uncommon complaints. The *rickety spine* is characterized by a wide curve, a flexible spine, the absence of angularity, while the kyphosis disappears when the child lies flat upon the face.

The *prognosis*, so far as the disease itself is concerned, is fairly favorable, but the intercurrent diseases may carry off the patient. Bronchitis, bronchopneumonia, phthisis, laryngeal spasms and convulsions are always to be feared. Anemia, emaciation and general debility are liable to be serious complications. While the disease has a tendency to progress toward an unfavorable termination by some complication, proper care, diet and the indicated remedies will prevent this in the majority of cases.

Treatment.—In caring for these cases it is necessary to give judicious thought to the *health of the mother* as well as the child. Frequently the mother has not sufficient health or strength to supply normal milk for her child. This occurs among those women whose environment is conducive to physical debility; prolonged nursing and too frequent pregnancies are many times the causes of a maternal debility which impairs the child's nourishment at a period when the osseous structure needs the most perfect nutrition. When it is clearly evident that the mother can not furnish the proper milk it will be necessary to employ an artificial food, the best of which is cow's milk. A judicious admixture of whole wheat or barley gruels will often furnish the salts which are lacking. A wet nurse may answer well in some cases, but the uncertainty as to her health may be a serious objection. The *feedings* must be regulated as to time as well as quantity. Too many mothers and nurses are inclined to feed the child every time it cries, and if the stools are constantly watched overfeeding may be detected by the presence of curds. Such a condition would indicate the necessity of less food, greater regularity in feeding and the use, perhaps, of barley water as a diluent and a diastase to aid the starchy digestion. As the *child grows older* equal care will be required in the adaptation of a mixed diet. At this period it is possible to receive much help from beef juice, the yolk of eggs, peptonized milk or beef peptonoids. Still later it will be possible to push the diet to the point of extreme nourishment if the stomach and intestine are able to perform the work. Since fats are usually deficient the food must supply them, as cream, butter and Russel's Emulsion. Daily rubs with cocoanut or olive oil or lard are of great value.

The *hygienic care* is of equal importance. Systematic bathing, a proper amount of pure air and clothing which insures perfect warmth, are essentials which should not be overlooked. The *bed* should be firm and yet comfortable, and the child should not be propped up with too many pillows; the decubitus must be frequently changed to obviate bony deformities, and the child should not be allowed to walk until the osseous structure is firm. To prevent this *splints* are often required and later mechanical appliances will be needed to obviate the tendency to spinal curvature. *Orthopedic attention* will often be demanded as the child develops when the deformities do not respond to the care already outlined. The digestive disturbances will generally respond to the use of *pepsin, hydrochloric acid* and the remedies usually indicated for these perversions.

The *medicinal treatment* is of paramount importance, and there are many remedies which are peculiarly indicated. *Phosphorus, calcium salts, iron* and *cod liver oil* have been standard therapeutic agents for a long time. The first named remedy has been used (grs. 1/200-1/100) three or four times daily, and it is also useful when combined with sweet or cod liver oil. The last named agent is still used satisfactorily by inunction. *Phosphorus,* in the third decimal potency, is one of our best homeopathic remedies. It pertains, in its symptomatology, not alone to the lack of bone development, but applies also to a general debility where emaciation is attended by marked nervous exhaustion. In combination with iron, as *ferrum phos.,* and in combination with calcarea, as *calcarea phos.,* it is most serviceable. The former we shall find indicated when there are symptoms of attending scorbutus or bronchitis. The latter form relates to the constitutional disturbances for which calcarea is particularly valuable; this peculiar cachexia is attended by the sweating, gastro-intestinal disorders, swollen glands and respiratory weakness. *Calcarea fluorica* will be indicated when the malnutrition is evidenced by a suppurative tendency and varicosity of the veins. *Calcarea sulph.* is called for in conditions of fibrosis and when the suppurative process is characterized by a thick mucous discharge. *Calcarea iodid* is useful when the glandular enlargements—particularly the tonsils—are prominent symptoms in the disease. *Phosphoric acid* will be the remedy when exhaustion has reached an extreme degree and particularly when the mental debility is first noticed. Such patients are listless, disinclined to move and there are frequent stools with much flatus. The maldevelopment will be manifested also by the dry and cracked lips, the bleeding gums and the swollen and dry tongue. In such cases hepatic and splenic enlargement are also prominent features. *China, picric acid, sepia* and *sulphur* may be compared.

Silicea is the next remedy of importance and it has many symptoms similar to those of the phosphorus and calcarea combinations. The child shows the effect of imperfect assimilation and defective nutrition, but it is more scrawny than the calcarea patient. The abdomen is large, the fontanelles are open, caries and necrosis are common and the complexion is of an ashy color. *Fluoric acid* and *sulphur* bear a close relation to both phosphorus and silicea. *Baryta carb.* is frequently indicated in scrofulous children of dwarfish appearance. The mentality is impaired, the glands are indurated, the abdomen is distended and hard, the teeth decay early, coryza is almost a constant symptom, tonsillitis is common, while the cold hands and clammy feet are always observed. *Kali iodid* should always be remembered when there is a syphilitic history and the same is true of *aurŭm chlorid, mercurius* and *cypridol.* Other remedies which demand consideration are *hepar sulph., belladonna, echinacea, baptisia, staphisagria, mezereum, lachesis* and *theridion.*

OSTEOMALACIA.

Osteomalacia, or mollites ossium, is a rare affection which is clinically characterized by a softening of the bones after their complete development; it is due to solution of the lime salts, the decalcification permitting a distortion or fracture of the bones. It is a rare disease and afflicts adults only. The cause is not known. Women are chiefly affected during the puerperal state and it seems to prevail in certain localities. Unhygienic surroundings, impure and insufficient food and senility are etiological factors.

Pathology.—The pathologic change first shows an *increased vascularity* in the bony structure. Solution of the tissues gradually ensues, in which there is a progressive disappearance of the lime salts. As this process takes place from within outward, the central cavity becomes enlarged and as the matrix gradually disappears the cortical portion may become very thin. The resultant fluid is mucoid in character, but as it mixes with the bone marrow it becomes thin and viscid. The *periosteum* becomes similarly vascular and eventually becomes thickened. Lactic acid is thought to be the solvent, inasmuch as it has been found in the urine and also in the medullary canal of these patients. The *extension of the pathological process* is uneven, the vertebrae, bones of the pelvis and thorax being the osseous structures most frequently involved. When the vertebrae are the seat of disturbance a backward and lateral curvature of the dorsal region (kyphoscoliosis) and a forward and lateral curvature of the lumbar region (lordoscoliosis) are observed, while at the same time we may observe an anterior protrusion in the cervical region. The sternum is unusually prominent and distorted while the thorax is compressed laterally. The same is true of the pelvis, which shows a deformity by the projection forward of the sacrum.

Symptoms.—*Pain* is the first symptom which is encountered, and it is experienced for the most part in the neck, back, thighs and sacral regions. It usually develops gradually, but when it is once established it is liable to be continuous. It is located in that part of the anatomy which furnishes the seat of first pathological development. Sometimes it partakes of the rheumatic character; it may be deep seated and aggravated at night or during exertion. The patient often assumes a hobbling gait on account of the suffering and, in many cases, the locomotion is so perverted that he is obliged to take to his bed. *Tenderness* over the involved part is a constant affliction and while in bed the pressure of the clothes is equally painful. *Fractures* are often observed, either in the ribs or extremities, and these lead to further distortion, inasmuch as the repair is not generally perfect. The *constriction of the chest* gives rise to dyspnea and other distressing thoracic symptoms. When the pelvis is involved in women it leads to pronounced deformities which are serious in case of labor. The bones, when affected by this disease, are often so soft that the limbs are bent out of place and this is a possible feature in all of the long bones, though the smaller ones are apparently exempt from the pathological process. *Fever* is an occasional symptom, though this is by no means constant. The *general health* is decidedly impaired in the later stages of the disease, but in the earlier stages this is not so marked.

Diagnosis.—The characteristic deformities will define the disease when it is well established. In the early stages the pain may be suggestive of a rheumatic or gouty attack. The peculiar hobbling gait is a distinctive feature and it should be readily distinguished from rachitis by the fact that it occurs always in adults and it is a degeneration of the fully developed osseous structure. Moreover it affects the shafts of bones rather than the epiphyses.

Prognosis.—Few cases recover and intercurrent diseases are liable to abbreviate the duration of life. The most unfortunate feature is the tendency toward a prolonged course, and death is generally due to exhaustion unless some complicating disease like pneumonia cuts short the suffering.

Treatment.—Not much is to be expected from treatment and the most a physician can do will be to *palliate the suffering* by remedies, and opiates if necessary. The *general care* should be much the same as that demanded in rachitis. The diet, exercise, out-of-door life and tonic treatment are requirements which must be considered. Therapeutic efforts have accomplished but little and though some claims are made as to the value of *phosphorus* as an internal medication, no satisfactory evidence of its efficiency has been recorded. The syrup of lactophosphate of lime and the syrup of the hypophosphites have been highly recommended. *Calcarea, iodin, iodid* *of lime, baryta iodid* and *arsenicum iodid* have been suggested, but there are no reliable records as to their attaining results.

SCORBUTUS.

This disease, otherwise known as scurvy, is a constitutional disorder which is created by a diet deficient in fresh vegetables and fruits. It is *characterized* by anemia, emaciation, marked debility, spongy gums, ecchymoses into the cutaneous structure, fibrous exudate into the deeper parts, the disintegration of tissue, and occasionally by internal or external hemorrhage of considerable extent. Few cases of scurvy are seen today, inasmuch as our preservation of vegetables has reached such a degree of perfection that it is possible to have this form of food under all circumstances. Protracted *absence at sea,* camp life and isolated conditions no longer furnish the possibilities of such epidemics of scurvy as have appeared in the past. As our physiologic and economic conditions improve there will be no reason for its existence at all. Where the crowded settlements are subject to sanitary imperfections and when the food is impure we may expect sporadic cases and sometimes epidemic outbreaks. It will always be necessary to carefully watch the food supply and hygienic conditions in our prisons, almshouses and all overcrowded quarters for errors in these respects always favor this disease. Testi and Beri have isolated a micro-organism which by inoculation in guinea pigs produced symptoms similar to those of scurvy. More conclusive evidence in this respect is desired.

Aside from the *absence* of *organic salts* obtained from vegetables, which occurs when this dietetic error exists, the free hydrochloric acid and the total acidity are reduced to such an extent that food is not properly peptonized. Thus a digestive disturbance is liable to be established which may prolong the debility even when the proper food is supplied. *All ages* are susceptible to the disease when the predisposing factors prevail and yet those of advanced years are more frequently affected. In addition to the unhygienic surroundings and the dietetic error, it must be remembered that *mental anxiety* and *nervous depression* favor its development.

Pathology.—The pathologic findings are more distinctly characteristic. The blood is thinner than normal, but leukocytosis is not present. *Subcutaneous hemorrhages* are manifested in the ecchymotic spots and a more pronounced hemorrhage will be found underneath the periosteum of the long bones, particularly the femur. *Articular bleedings* are often in evidence and the same is true of the muscles, serous membranes and internal organs. The *intestinal mucous membrane* may be the seat of ulceration, while the gums are spongy and bleed easily and the teeth are subject to decay. The *spleen* is soft and enlarged, while the heart, liver and kidney present a fatty or parenchymatous degeneration.

Symptoms.—The disease is *insidious* in its development, though in exceptional instances the onset is sudden and the progress toward a fatal end is rapid. *Debility* and *emaciation* gradually increase, while the mental and nervous irritability may be unusually distressing. The appearance of the patient is peculiar, for the eyes are swollen and the face is bloated, pale and often the color simulates a bruised condition. The *skin* is dry, poorly nourished and dequamates easily upon friction. Small petechial spots, reddish or brownish in color, may appear a little later upon the limbs, trunk and face. The patient will often complain of nocturnal pains and dyspnea from the slightest exertion.

Swollen gums, which later become spongy and bleed freely, is one of the first characteristic symptoms. The teeth become loose and may drop out, the breath is offensive, the tongue swells and the mucous membrane of the mouth may be the seat of small hemorrhages. The appetite is impaired and obstinate constipation may be followed by a debilitating diarrhea. *Scorbutic dysentery* is a frequent feature and while the urinary changes are not marked, albumin may be discovered and in exceptional cases a grave form of nephritis may exist. The skin, following the petechiae and ecchymoses, may assume a muddy color or it may present yellowish brown patches. As the *hemorrhage* from the free surfaces generally starts in the region of the hair follicles, the nutrition of the hair in these parts is impaired and it may lose its color or drop out. The extravasated blood may be sufficient to form papules on the skin, or vesicles may be formed and if these rupture, bloody crusts develop over an ulcerating and discharging base. The *subcutaneous hemorrhages* may be large enough to establish an extensive area of ulceration and to a certain extent interfere with nutrition by subsequent atrophy and sclerosis. *Secondary inflammation* in these parts is liable to be followed by suppuration, deep-seated ulcers and gangrenous conditions. Pain invariably attends these extreme pathological changes as the result of pressure or necrosis. *Subperiosteal effusions* not only cause excessive pain, but sensitive nodules may be felt along the bony shaft. When the articulations are implicated the pain is accompanied by swelling, and the suppuration may develop such destructive changes that ankylosis may result. *Epistaxis* is not uncommon and when hemorrhage into viscera occurs hematuria, hematemesis, or hemoptysis may occur, or secondary nephritis, gastritis or pneumonia may develop.

The *heart action* is usually feeble and upon exertion palpitation, arrythmia and often hemic murmurs may be detected. As the blood vessels are diminished in caliber cardiac dilatation is not uncommon and the pulse will be soft and small. When the debility increases the *nervous irritation* will be more pronounced so that insomnia and delirium are possible features. In rare instances *meningeal hemorrhage* will be a complication. *Embolism* of the lungs or spleen, with resulting infarction, may be the cause of a sudden death. The weight usually decreases with the progressive debility, but there are exceptions in which the patient retains a fair amount of adipose. The temperature is generally subnormal in well marked cases, but if the viscera are involved sufficiently to create inflammation or suppuration, a rise of temperature may be expected.

Diagnosis.—The infrequency of the disease is the greatest excuse for diagnostic error. *Purpura* will be difficult to differentiate in certain instances particularly when the hemorrhagic spots are limited to the lower extremities and are unattended by hemorrhage into the deeper tissues or from the mucous surfaces. *Mercurial stomatitis* may give simulating symptoms in the mouth, but the history and other characteristic signs are quite distinctive.

Prognosis.—The prognosis is invariably favorable in cases not far advanced when the correct diet is instituted. When complicated by hemorrhagic invasions of the internal organs or when embolic infractions exist, we should not expect a favorable result from our treatment. When the osseous or other tissues are involved by syphilis or when other infectious diseases attend, the prognosis is always grave.

Treatment.—The first essential of treatment is *prophylaxis,* and this is attended to when the normal mixed diet is obtained. Such fresh vegetables as potatoes, onions, sauerkraut and fruits are the best antiscorbutic foods, and these alone will cure the average incipient case. *Fruit juices* have obviated the danger of scorbutus which has heretofore existed in both naval and army life. *Meats* should only be sparingly permitted until there are positive signs of improvement. *Milk,* eggs and farinaceous dishes are a good compromise as we approach the proteid diet. The mouth should be cared for by proper antiseptic solutions, the ulceration should be treated surgically, and the general symptomatology of the patient should be watched and cared for with relevancy to complications or serious conditions.

Internal medication does not accomplish so much as the dietetic regulations, though many times there are special symptoms which are indicative of a similimum. Hemorrhagic remedies like *secale, nitric acid, hamamelis, ipecac* and *china* are often of service and may be called for by the totality of symptoms. *Phosphorus, ferrum phos., phosphoric acid* and *terebinth* are quite frequently useful, as they relate to tissue metamorphosis, hemorrhages, anemia and digestive disturbance. Often the heart needs the assistance of a cardiac tonic, but frequently perversions of this organ are corrected by such remedies as *crotalus horridus, naja, lachesis* or *arsenicum.* Should women be affected and uterine hemorrhage be a symptom, then we may rely upon such remedies as *cimicifuga, pulsatilla, sabina, trillium, millefolium* and *erigeron.* In case of hematuria *mercurius, hydrangea, berberis, chimaphila, terebinth, uva ursi, vesicaria* and *sabul serrulata* may be required. When hematemesis is a symptom *hydrastin hydrochlorate, gallic acid, secale* and *ipecac* are usually indicated, and when there are complicating conditions of the lungs *creosote* and other remedies which relate to pneumonia or tuberculosis should be studied.

INFANTILE SCORBUTUS.

This is a form of scurvy which is peculiar to infants and is characterized by the additional evidences of rachitis. It is found most commonly among those infants reared upon *artificial foods,* and it is not confined entirely to the children of poor parentage; it is quite frequently discovered in the offspring of those who have suffered from debility incident to luxury. It occurs generally during the *first two years of life* and between the seventh and fourteenth months most frequently. The nature of the disease is not well understood. It has been called "Barlow's Disease" on account of the exhaustive research made by this author, and it has also been termed "periosteal cachexia" from the subperiosteal hemorrhage which characterizes its pathology, while some observers still consider it as a phase of acute rachitis. It is of *rapid development* and the pathologic features, in addition to the subperiosteal hemorrhage, are characterized by a thickened and extremely sensitive condition of the bones, with a marked tendency to softening between the epiphysis and shaft, and occasional intermuscular extravasation. The lesions of rickets are also added. The lower extremities are most frequently affected, but the forearm, scapula, vault of the cranium and face are affected in exceptional cases.

Symptoms.—*Impaired nutrition* is usually in evidence, though the signs of wasting are not always present. The *skin* has a dark or muddy appearance similar to that when the adult is affected. *Pain* is a decided factor and it will be noticed by the child's crying whenever he is moved or the limbs are touched. *Swelling* of the limbs, as the result of the subperiosteal hemorrhage, soon develops symmetrically, though one limb is liable to be larger than the other. The parts are shiny and tense, though there is rarely any pitting upon pressure. In some cases there may be a separation of the epiphyses, while the limbs are flexed at first but later are straightened or everted. *Other bones* are involved as the disease progresses. The joints are rarely affected. The sternum, adjacent cartilages and a part of the contiguous ribs may be crowded in as if affected by violent pressure from without. Barlow describes a peculiar puffing with slight staining of one upper eyelid, while the other is soon similarly affected. Hemorrhages from the conjunctivae and mucous surfaces are often present. *Ecchymotic* spots may also be observed on the skin; while the gums swell and become spongy. The *temperature* may be subnormal, normal or fairly high (102° F.). *Anemia* is liable to be a marked feature.

Diagnosis.—The diagnosis is generally accepted by the differentiations of Barlow as follows: The predominence of lower limb affection, with the immobility which may lead to pseudo-paralysis; the excessive tenderness with swelling of the lower limbs; the shiny and tense skin which presents little local heat and no pitting; the thickened bony shafts after the swelling subsides and the fracture which may occur near the epiphysis together with the swelling of the gums about the teeth only and minute transient ecchymoses.

Prognosis.—The prognosis may be considered as unusually favorable if the proper dietetic treatment is instituted.

Treatment.—The treatment which has been outlined under scorbutus will generally apply in these cases. The correct diet will be the most difficult to establish, though the modified cow's milk, with the additional employment of meat juices and orange or lemon juice, will greatly facilitate the recovery. Orange juice freely administered is especially valuable. Hygienic care is of paramount importance and such remedies as *calcarea phos., ferrum phos., phosphoric acid, phosphorus* and the *hypophosphites* are usually indicated. *Arsenicum, iron* and *strychnia* may be demanded as tonic remedies. The limbs may need splints in case of fracture.

PURPURA.

Purpura is an expression of a constitutional condition, characterized by extravasations of blood into the skin irrespective of traumatism. The purpuric spots are designated as "petechiae" when they are small, and as "ecchymoses" when they are large. At their first appearance they are deep red in color, but later become darker and gradually fade into brownish-yellow spots. Pressure does not cause them to disappear. The time required for coagulation may be estimated by the coagulometer. While normal blood will clot in three to five minutes, purpuric blood will require ten to fifteen minutes. For the convenience of study the disease is classified under the following heads: Secondary or symptomatic purpura and primary or idiopathic purpura.

Secondary or Symptomatic Purpura.—Under this head we may first consider that form which is classed as *infectious.* The hemorrhagic spots may take the form of ecchymoses as observed in pyemia, septicemia and malignant endocarditis, or they may appear as a rash which, in its various forms, characterizes cerebro-spinal meningitis, typhus, measles, scarlet fever or smallpox. A *toxic variety* may be recognized as the result of snake virus and the action of such drugs as quinin, copaiba, belladonna, mercury, ergot and the iodids. *Cachetic purpura* is occasionally recognized in association with the constitutional disturbances accompanying cancer, tuberculosis, leukemia, pernicious anemia, nephritis, scurvy and often as a feature of senility. The purpuric spots are usually confined to the lower limbs in these instances. *Neurotic purpura* has been recognized in connection with such nervous diseases as tabes, following an attack of fulgurating pains; in such instances the hemorrhagic evidence is manifest at the point of severest pain. Purpuric spots may also accompany myelitis, hysteria and neuralgia. *Mechanical purpura* is a rare sequence of venous stasis or it may attend the paroxysms of whooping cough and the convulsions of epilepsy. All of these forms of purpura may be regarded as secondary to some other disease.

Primary or Idiopathic Purpura.—This is recognized either as simple purpura, arthritic purpura or purpura hemorrhagica. *Simple purpura* is a mild form occurring most frequently in children and appearing commonly upon the legs, with occasional involvement of the trunk and arms. The extravasations may appear as petechiae, ecchymoses or blood streaks (vibices), and often in crops. They are gradually transformed into brownish-yellow spots and finally disappear. Purpura simplex is regarded by some as rheumatic in character, though arthritic manifestations are not commonly observed. Fever is not usually a symptom and the disease is not generally protracted beyond a week or ten days.

Arthritic purpura or Schönlein's disease, is characterized by polyarthritis and an eruption varying from the purpuric spots to an urticaria or an exudative erythema. It occurs most frequently in males at middle life, though it is not confined to that sex. It is ushered in by prodromal symptoms of headache, articular pains, slight anginal attacks, sore throat and a fever which reaches as high as 103°-104° F. The joints are soon swollen, painful and decidedly tender. The eruption is confined mostly to the legs and the joints affected. When there is a combination of wheals and purpura it is defined as *purpura* *urticans* and when vesication occurs it is termed *pemphigoid purpura;* again the eruption may consist of these different types combined. Edema is occasionally pronounced and is excessive on the chin, lower lip and eyelids, the purpuric spots being in evidence where the edema is extreme; fever is invariably present when edema exists. Hemorrhage from mucous membranes is not common, epistaxis being of most frequent occurrence. The urine is often reduced in amount and may contain albumin. The *origin* of the disease is not clearly established as rheumatic, though with the symptoms of endocarditis present this theory is quite tenable. It has the features of rheumatic fever plus the purpuric manifestations. The *diagnosis* presents no difficulties when the polyarthritis, purpuric rash and edema are present. The *prognosis* is invariably favorable unless unexpected complications appear. There is always a marked tendency toward relapse or recurrence.

Henoch's purpura is a form of purpura which occurs chiefly among children and is characterized by its tendency to relapse or recurrence, by a cutaneous eruption which simulates erythema multiforme rather than purpura, and gastro-intestinal crises with vomiting and diarrhea. Painful and occasionally swollen joints, localized edema of the skin, and hemorrhages from the mucous membranes are also characteristic symptoms. When the hemorrhage takes place into the kidney a severe hemorrhagic nephritis may follow. The above symptoms are not always constant and the intestinal crises may often exist without any suggestion of the correct diagnosis until hemorrhage appears. In many respects it simulates angioneurotic edema.

Purpura hemorrhagica, also known as morbus maculosus of Werlhof, is the severest form of purpura. Though its etiological history is not fully established it is believed to have some relation to infectious diseases, particularly rheumatism and malaria, and it most often exists in association with a state of debility. Females are predisposed to it more than males, and though it may appear at any age, it is more liable to appear during middle or comparatively early life. The *symptoms* are announced by a prodromal malaise with slight headache, anorexia and exhaustion. After this debility has existed for a short time a moderate fever appears, followed later by the appearance of purpuric spots on the skin which rapidly increase in number and size. *Bleeding* from the mucous surfaces soon takes place and it may occur as epistaxis, hematuria or hemoptysis, with a marked resultant anemia. The prostration may be prolonged and associated with pains in the limbs, back and abdomen, or it may be intermittent, a recurrent malaise usually presaging another hemorrhage. Death may result from the loss of blood or apoplexy. The favorable cases run a course of ten days or two weeks, while a malignant form, *purpura fulminans,* is liable to be fatal in twenty-four hours. This form is generally observed in children and is characterized by cutaneous hemorrhages which develop rapidly, though in some cases death may come before the hemorrhage.

Diagnosis and Prognosis.—The *diagnosis* is usually made without difficulty and the only confusion is liable to occur in the case of scurvy. This disease, however, should be suspected from the dietetic cause and the characteristic spongy condition of the gums, the brawny indurations and the more scanty hemorrhage. The *prognosis* is favorable in the mild cases and recovery usually takes place in a week or ten days. The severe cases recover slowly, while the fulminating cases terminate unfavorably in twenty-four hours.

Treatment.—The treatment consists in the use of such measures and remedies as will correct the debility and hemorrhage. The usual tonics and dietetic recommendations have been employed and with generally beneficial results. *Arsenic,* without doubt, has given the most encouragement and this is not by any means without reason, for its hematic value in anemia is undisputed. Fowler's solution and combinations with iron and strychnia are the most popular forms of its employment. In the rheumatic form of the disease *sodium salicylate* has been recommended, but it does not give the satisfactory results as in rheumatic fever inasmuch as that relation is not clearly established in this disease.

For the *hemorrhage* the usual astringent remedies have been advised. Of these those most frequently used are *ergot* (minims 5-30), *persulphate of iron* (grs. ¼-½), and *dilute aromatic sulphuric acid* (minims 5-15). *Turpentine, lead acetate, tannic* and *gallic acid* have also been employed to check the bleeding. *Calcium chlorid* (grs. 5-30) four times daily, has been used to increase the coagulability of the blood. The internal and subcutaneous administration of the *suprarenal extract* (minims 5-10) has, however, almost entirely supplanted the older emergency remedies. A solution of *gelatin* (2 per cent) in normal salt solution has been given subcutaneously and per rectum with decided success.

Among the homeopathic remedies *nitric acid, terebinth, china, hamamelis, hydrastis* and *secale* are of first importance so far as hemorrhage is concerned. *Phosphorus, ferrum phos, phosphoric acid and picric acid* are next in value. When chronic cardiac conditions are underlying factors we may find help from such remedies as *lachesis, crotalus, naja, spigelia* and *kalmia.* When the urticarial conditions are prominent, *natrum mur, graphites, pulsatilla, calcarea, sepia* and *sulphur* may be demanded. *Kali iodid, arsenicum iodid, mercurius, mezereum, phytolacca, rhus tox, anacardium, urtica urens* and *apis* will correspond to the totality of symptoms in a large class of cases.

HEMORRHAGIC DISEASES OF THE NEWBORN.

Syphilis Hemorrhagica Neonatorum.—When this disease exists cutaneous extravasations and bleeding from the bowels, kidneys or from the umbilicus may be discovered shortly after birth. The skin is deeply jaundiced, the stomach rejects food, emaciation is pronounced and death usually occurs in the course of a week or ten days. The autopsy reveals syphilitic lesions in the liver, lungs and other viscera.

Epidemic Hemoglobinuria.—This was first described by Winckel in 1879 as an epidemic form of hemorrhage occurring most frequently in lying-in institutions. It is extremely fatal and usually appears about the fourth day, or often as early as the second day, after birth. The skin becomes icteric, while fever, rapid respiration, occasional cyanosis, vomiting and diarrhea are attending symptoms. The *urine* contains albumin, methemoglobin and a deposit of epithelium and tube casts. The *blood* has an excess of leukocytes and granular detritus. *Hemorrhages* may occur in the kidney and other organs as well as in the mucous membranes. An infectious origin is presumed. The autopsy discloses a staining of the skin and internal organs with numerous punctiform hemorrhages, the spleen is enlarged, the kidneys are dark brown in color and the tubules are filled with granular debris. The liver, heart and other viscera show some fatty degeneration.

Acute Degeneration of the Internal Organs.—This is known as Buhl's disease and is similar to that just described as Winckel's disease. It is, no doubt, an infectious disease of the newborn, characterized by cyanosis, jaundice and visceral hemorrhages, with an acute fatty degeneration of the internal organs.

Morbus Maculosus Neonatorum.—This is a form of bleeding which takes place during the first week or, in exceptional cases, two or three weeks after birth. The hemorrhage usually takes place from the bowels (melena neonatorum) though it may come from the mouth, nose, stomach or navel. *Hematogenous jaundice* is occasionally observed and fever is frequently present. *Cerebral hemorrhages* and infections are regarded as causes. Postmortem changes are not marked, though ulceration of the esophagus, stomach and duodenum have been found. The disease is usually fatal, death occurring in from one to several days.

Treatment.—So far as remedies are concerned, little in the line of treatment will avail. Complete rest is necessary and for this purpose it will often be best to feed the child with a spoon or bottle instead of permitting it to nurse. Warmth must be consistently maintained and for this purpose the use of the incubator has been of advantage in recent practice. *Gallic acid* and the indicated homeopathic remedies may suffice in cases where they may be administered in solution. It is often necessary to control the hemorrhage by means of hypodermatic injections of ergotin in minim doses every six hours.

HEMOPHILIA.

This is a constitutional disease which is characterized by uncontrollable bleeding which occurs spontaneously or from traumatism. It is hereditary and is often associated with some other disease, particularly arthritis. That it is hereditary must be admitted inasmuch as we so frequently find a family tendency toward bleeding. The transmission of the disease seems to occur through the female line, though the direct male descendant is most liable to the affliction. Blondes seem to be most frequently predisposed.

Pathology.—The nature of the disease is unknown, and there are no special pathological features which are distinctive. The pathology is found in the arterioles, whose middle muscular tunic is often absent or very thin. *Vaso-motor disturbances* must be taken into consideration inasmuch as emotional excitement is frequently a factor. *Hemorrhages* into the joints, with subsequent inflammation of the synovial membranes, are occasionally discovered. The blood changes are not marked, a diminution in the number of leukocytes and retarded coagulation must be regarded as pathologic features which favor the disease.

Symptoms.—The disease is first manifested by the occurrence of a persistent *hemorrhage* from slight wounds. This may take place following a skin-scratch, the extraction of a tooth, or from a slight surgical operation. It may occur at birth following the cutting of the cord. The *spontaneous hemorrhages* usually take place in the form of nosebleed after blowing the nose, or it may come from the mouth, stomach or superficial wounds. *Petechiae* occur from trivial causes and ecchymoses or "black and blue" spots follow the slightest injuries. Extensive *extravasations* (hematoma) may result from contusions. *Menstruation* is liable to be profuse in female subjects, though childbearing is not generally attended by an unusual loss of blood. *External hemorrhages,* particularly those of the nose and mouth, are inclined to be severe and difficult to check. The patient invariably reacts promptly when the hemorrhage is stopped, though repeated loss of blood may develop a profound anemia. *Arthritic symptoms* may arise from hemorrhage into the larger joints and the febrile condition which arises may simulate rheumatic fever. In some instances the joint pains may presage an attack of hemorrhage.

Diagnosis.—When the hereditary influence is established or when the hemorrhage takes place frequently from slight injuries we may suspect hemophilia. *Peliosis rheumatica* is a simulating disease, and should be diagnosed by the presence of edema urticaria and its distressing itching.

Prognosis.—The tendency is toward a fatal termination in case of repeated hemorrhages. There is a possibility of the patient outgrowing the disease with advancing years. The fatal cases are almost invariably found in the young.

Treatment.—The *prevention* of hemorrhage is paramount.

Such patients should be on constant guard against traumatism and surgical operations should not be performed on such subjects, unless it is absolutely necessary. It may be well also to discourage marriage in the case of women afflicted with hemophilia for fear of abortions and a possible fatal hemorrhage at confinement. Anders, however, quoting Kolster, says of 130 cases of pregnancy and labor, the death of the mother occurred in only three and abortion in sixteen cases. *Hygienic measures* should be instituted and the nutrition must be brought to the highest standard. *Outdoor exercise* and a general development of a normal physical resistance will often thwart the tendency to bleeding as the patient grows older.

During an attack quiet is necessary, while styptics and internal remedies, which have been previously mentioned under purpura should be employed in case of hemorrhage. For styptics perchlorid of iron, at first diluted and later in full strength, may be relied upon. Tannic acid in saturate solution or as a powder is a valuable agent and it may be applied on cotton or gauze whenever a plug for cavities is needed. Lint saturated with spirits of turpentine is also serviceable in many instances. In epistaxis ice is often the only required application and if this does not suffice, plugging will be necessary. Solutions of suprarenal extract are now the most convenient and efficacious agent for treatment in case of hemorrhage. Their hypodermatic administration may be demanded.

The *internal remedies* have been previously mentioned with reference to purpura, but here the *suprarenal* extract grs. 2 or adrenalin (m. 5-30) every three hours, gives the most positive relief. One dram of adrenalin chlorid may be added to half a pint of normal salt solution and used by hypodermoclysis or intravenous injection. It may also be used hypodermatically. *Hydrastis* fluid extract (gtt. 5-15) may be used every three hours, or *hydrastin hydrochlorate,* in the first decimal potency. *Calcium salts* are also to be considered. The convalescence should be treated in accordance with the demands of the case and the indications for remedies.

OBESITY.

This disease is included under constitutional diseases for the want of a better classification. In some people a considerable degree of obesity is a normal condition, while in others the deposition of fat in excess is a manifestation of disease or a perverted function. Thus the term obesity or adiposity should be used to describe a state in which there is some suffering or impairment of the physical health from this cause. When it is an inherited condition little can be done to prevent it and no apparent harm results with the exception that the sufferer is incapacitated from taking a sufficient amount of exercise. Where it develops from over eating, alcoholic stimulation and a lack of physical exercise, it is an acquired disease and leads to a disordered metabolism which favors organic disease. Obesity sometimes follows an infectious disease or it may appear in women after the menopause. In a large class of people the increase of fat is a natural process after the fortieth year. Another class of patients, to my mind, become obese as the result of faulty or deficient elimination.

The *pathologic* condition is characterized by the marked increase of fat deposited throughout the body with an especially large amount distributed over the abdomen in most cases. This excess fat is not distributed under the skin alone, but the internal viscera suffer from its presence as well. The heart is overlaid with it and the intermuscular tissue may suffer from a fatty infiltration. The arteries also are affected, the changes being noted in the intima and media, while chronic endarteritis with sclerosis and thickening of the vessels may take place. The veins may also become varicose from this cause. The blood is increased in specific gravity and in many cases the hemoglobin percentage is also increased. The lungs may suffer from passive congestion or edema on account of the cardiac weakness. The liver is affected similarly, the kidneys may become infiltrated and the stomach may be dilated on account of the fatty invasion of its muscular stroma; a gastro-intestinal catarrh is not a remote possibility from this cause, and its prolonged existence may favor gastrectasis.

Symptoms.—Aside from the gross appearance caused by obesity the cumbersome weight is liable to cause *dyspnea* from the slightest exertion. This exists not alone from the weight but is dependent upon the abnormal amount of fat in the abdomen and chest which acts as a mechanical disturbance of respiration and cardiac action. On account of the increase in the vascular network in the obese the heart has to force the blood through a greater number of blood vessels; hence the fatty heart of these patients means more than the superimposed fat and the condition of infiltration for such a state favors other organic changes. *Passive congestions* are liable to exist in other organs when the fatty change involves the arterial system. This accounts for many of the associate affections of the liver, lungs and digestive tract. *Anemia* is a natural sequence in this condition, the skin being pale while the muscles become flabby. Such patients can not endure work or exercise without marked fatigue. In women it often explains the uterine disorders both functional and organic. Another point of importance is the fact that obese patients do not well endure an acute infection and functional derangements of all kinds are liable to be exaggerated. Digestion is easily disturbed, the sexual power is debilitated, constipation is common and the mind is not normally active. The skin function may often be impaired and in addition to the common excoriations, pruritus, acne and eczema are occasional perversions. *Profuse sweating* is a common complaint of the obese, and if the skin and kidney functions are not active, polyuria or oliguria may be experienced. The *pulse* is usually small and rapid while the heart sounds are diminished in intensity.

Treatment.—The principle of treatment is to obviate as far as possible the further increase of fat. This will require a prophylaxis which shall consider the hereditary predisposition and the diet. The former is corrected somewhat by a thoughtful anticipation of hereditary tendencies and the latter will be attended to by a painstaking elimination of fat-forming foods. The tendency for such patients to overeat will demand close watching. Sugars, fats, starches and alcohol must be restricted in the majority of cases, though in a purely hydremic form of obesity albuminoids, hydrocarbons and fat producing materials may be required for a time at least. Severe measures employed to reduce fat should not be tolerated, and to this end it will often be necessary to seriously caution patients against many of the advertised remedies. Water is generally used in abundance by fat people and this habit may be restricted with due regard for the system's demands.

Physical exercise is an adjuvant form of treatment which must not be overlooked. This, however, must be taken with a proper consideration for the patient's ability to withstand extreme physical strain. Oxidation is naturally increased by exercise, but it is not successfully obtained by forced exercise. Myocardial degeneration and cardiac dilatation are so commonly present that they naturally contraindicate any extreme measure of this kind. *Baths* which favor skin elimination are necessary expedients of treatment, but these also must be undertaken with due regard for the possible resultant depression. The Turkish bath and the prolonged hot bath should not be tolerated. A cool shower taken with moderation is preferable. *Diuresis* and *catharsis* should be induced with equal caution lest sudden and unnatural depletion thwart the object of treatment. Massage and carefully applied gymnastics will often accomplish much for these cases. All measures which increase the elimination are indispensable in the treatment of this condition.

The *medicinal treatment* does not offer much encouragement except as it pertains to the proper indications and the use of remedies which favor the reduction of fat. *Phytolacca* has for a long time been regarded as a useful remedy for this purpose. Its action upon the glandular and fibrous tissues is well known and it is possible that it may have something to do with the reduction of fat. It has been used freely in the form of a watery decoction obtained from the berries. Gouty conditions must also receive proper consideration according to suggestions given under that subject. It is safe to say that any remedy which favors elimination without secondary depression or helps in restoring any perverted function will be useful in the treatment of this disease. *Thyroid* extract has also been recommended, but this refers only to that form of obesity which pertains to myxedema. *Phosphorus, calcarea carb., sulphur, silicea* and *mercurius* have also been suggested for favorable consideration.

DISEASES OF THE BLOOD AND DUCTLESS GLANDS.

ANEMIA.

Anemia is the pathologic condition in which the blood is deficient either in quantity or in one or more of its essential constituents. The disease is divided for study into two groups —primary or essential, and secondary or symptomatic anemia. In the former the blood or blood-making organs alone are implicated; in the latter, acute or chronic diseases, hemorrhage, etc., are the essential etiologic factors. Often the two forms are combined.

Symptomatic or Secondary Anemia.—This form being expressive of some existing disease, it is necessary to ascertain what disease or diseases it depends upon in order to intelligently deal with it. The following are the more important groups of secondary anemias:

Anemia Due to Hemorrhage.—This may result from hemorrhage from any source, uterine, gastric, intestinal and pulmonary hemorrhage and the rupture of an aneurism being the most common examples under this head. In like manner the cause may be found in a hemorrhage produced by a parasitic invasion of the intestinal canal or the presence of the distoma hematobium in the kidney. Epistaxis, menorrhagia and bleeding hemorrhoids are sometimes the traceable causes. In hemorrhages which are not fatal the process of regeneration occurs with great rapidity, and the normal amount of blood is re-established in a few days. Except in extreme conditions, absorption of the watery and saline constituents takes place quickly from the gastro-intestinal tract, while the albuminous elements are renewed more slowly; in a few weeks or months the red corpuscles approximate their normal count, while the hemoglobin is replenished more slowly. When the regenerative process is long delayed a pronounced anemia exists.

Anemia Due to Malignant Diseases.—Both carcinoma and sarcoma produce marked decrease not only of the hemoglobin but of the red-blood cells as well. This is true when the tumor is sufficiently large to produce clinical symptoms. Large sarcomas are more distinctive of blood constituents than are large carcinomas. The reverse is true in the case of small tumors. The vast majority of malignant tumors cause a well developed leukocytosis with an increase of the fibrin. The blood picture caused by malignant growths closely resembles that of suppuration but differs in having a greater degree of anemia, a smaller leukocytosis and fibrin frequently normal.

Anemias Due to the Drain of Chronic Diseases.—This class of anemias are found when there is a prolonged wasting of the albuminous elements of the blood. It is observed in chronic suppuration, nephritis, chronic dysentery, prolonged lactation and in connection with rapidly developing tumors. The anemias of malaria may be included in this group. Though the blood picture is that of anemia it is modified by the causative disease.

Anemias from Inanition.—This form of anemia is a result of starvation. It may exist because of gastritis or gastric ulcer which prevent the proper digestion of food, or it may be due to an interference with the ingestion and assimilation of food resulting from esophageal and gastric constriction by cancer. In these states the blood plasma is reduced more than the corpuscles, which are sometimes relatively increased.

Toxic Anemias.—These are induced by the action of certain poisons upon the blood, of which the most common are lead, mercury, arsenic and phosphorus. Such diseases as syphilis, malaria, tuberculosis, chronic nephritis and some of the infectious diseases also cause anemia by their toxic action on the blood. We may add to these the toxic influences which come from insanitary surroundings. The anemia of tuberculosis is a marked illustration of the toxic form and a similar condition may be the result of intestinal parasites.

Symptoms.—*Paleness of the skin* is probably the first symptom which directs our attention to a secondary anemia. This sign, however, is not always a positive evidence of anemia, inasmuch as it may be an idiosyncrasy without special significance. *Exhaustion, dyspnea, faintness* and *cardiac palpitation* are also suggestive. The most positive indications are found by blood examination. This will reveal a reduction in corpuscles and hemoglobin proportionate to the extent of the anemia—the hemoglobin showing the most distinct loss. The red corpuscles are pale and reduced in size, while there is often a moderate poikilocytosis. Nucleated red corpuscles, normoblasts and microblasts appear if the anemia is very severe.

The white corpuscles: The polymorphonuclear neutrophiles are moderately increased except in secondary anemias due to inanition, tuberculosis, syphilis, chronic nephritis, malaria and typhoid fever. In these diseases the count of the white cells is decreased, and the lymphocytes are relatively increased.

Diagnosis.—In making a diagnosis it will be necessary to compare this with *primary anemia.* It is always a condition secondary to some other disease, while the idiopathic form is a primary disease of the blood and the blood making organs. This may occur at any age, while primary anemia is prone to middle or advanced life. It furnishes a history of the causative disease mentioned, while primary anemia gives no such relation. It does not manifest such marked blood changes as are found in the idiopathic form, having a moderate reduction in corpuscles and hemoglobin compared to the marked reduction in the latter. The general symptoms relate more directly to the causative disease, while in the latter the anemia is more characteristic of blood changes. In the secondary anemia the gravity depends upon the primary disease, while in the idiopathic form the gravity depends upon the type of the blood changes and the progressiveness of the disease. Secondary anemia usually responds to the treatment and is often of short duration, while essential anemia is rarely curable except when it is of the chlorotic type.

Prognosis.—This will depend upon the cause of the anemia and the general condition of the patient. The majority of cases will respond promptly to proper treatment and the removal of the cause.

Treatment.—The removal of the cause will demand the treatment indicated by the symptomatology. This will be usually accomplished by such remedies as *china, china arsenicum, arsenicum iodid, ferrum phos., phosphorus* and *phosphoric acid. Iron* in full doses is often required. Saline transfusions may be of value. Nourishing food is most essential, while hygienic care and outdoor life are necessary.

PRIMARY OR ESSENTIAL ANEMIA.

This form of anemia has its pathological origin in the blood or the blood-forming organs, and diseased condition of other tissues is not presumed. Under this head we may include chlorosis, pernicious anemia, leukemia, pseudo-leukemia and splenic anemia.

CHLOROSIS.

This is an anemia often known as green sickness or chloremia, which is observed most frequently in young women. It is characterized by a marked relative diminution of the hemoglobin of the red corpuscles, which is no doubt due to a defective blood production.

Etiology.—Girls are affected more frequently about the time of puberty, scanty or absent menstruation being often the first suggestion. It may be seen in older women, the ages between twenty and thirty being the most prolific; when it is found in women beyond thirty it is spoken of as *chlorosis tarda.* It is supposed to be most common in blondes, though it is questionable if this is true. The most unfortunate feature is the fact that there is a tendency to recurrence from any cause affecting the general health. Men are rarely afflicted, though it is claimed that those of the male sex who have the habits and occupations of women, particularly tailors, are liable to suffer from the disease. It is rarely discovered in boys.

Heredity is a decided factor, as chlorosis may often be traced through the female members of a family. The inheritance of a delicate physical organization, not to mention the tuberculous and other taints which are handed down, naturally predispose. The lack of proper *hygienic conditions,* unhealthy habits, filth and poor nutrition always contribute favorably to its development. *Overwork* and under-rest, late hours and late dinners, insufficient and improper food, irregular hours for work or for meals, lack of healthy exercise and occupation, stimulants like coffee, tea, wine and cigarettes, are a few of the predisposing agents which develop the disease. It is necessary always to add to these causes the exhaustion which arises from digestive impairment or neurasthenia, from prolonged lactation and profuse menstruation, as well as from sexual excesses and masturbation. *Emotional excitement,* despondency, hysteria and mental perversions have their part in producing it, while grief, homesickness or disappointment in love, are always etiological possibilities. The change of climate and particularly a trip across the ocean is often the primary cause.

Sir Andrew Clark was the first to suggest that it was really a condition of copremia, a toxemia resulting from the colon absorption of ptomains and leukomains, inasmuch as these interfere with the hemoglobin without any destructive action upon the corpuscle. It was the theory of Bunge that the disease might arise from intestinal putrefaction due to imperfect stomach digestion, both of which views would seem to establish a relation between chlorosis and pernicious anemia.

Two suggestions are coming prominently to the front relative to the cause of chlorosis. First it is due either to imperfect development of the blood forming organs or their greatly increased burden due to the increased growth of the special organs at the time of puberty. Second the absence of an ovarian secretion is thought to be a factor in the underdevelopment of these blood forming organs.

Pathology.—Aside from the blood changes which are defined under the symptomatology we shall be able to detect little definite pathology. Virchow has mentioned arrested development of the aorta and large blood vessels as well as the hypoplasia of the uterus and adnexa. Abnormalities of the genitalia have also been found, but these conditions are apparently coincidents in the physical development rather than features of morbid anatomy. Myocarditis with cardiac hypertrophy and dilatation is sometimes observed.

Symptoms.—The first symptoms of the disease are not always indicative of the anemic condition which follows. The girl may show some signs of *irritability, nervousness,* depression of spirits and strength, languor and a disposition to be quiet. As the anemia progresses she will manifest symptoms of a more pronounced fatigue upon the slightest exertion. *Shortness of breath,* cardiac palpitation and dyspnea are noticed a little later. *Headache,* digestive disturbances and constipation are soon experienced, while scanty and irregular menstruation is a most typical manifestation of the approaching disease. *The gastro-intestinal features* are further marked by the perverted and capricious appetite, as the patient invariably cares for little food or else there is a craving for abnormal things, such as chalk, slate pencils, earth or sand, sour things, etc. The *tongue is pale and flabby* and often shows the indentations of the teeth. *Vomiting* may occur every morning and gaseous eructations and flatulency are very distressing. *Diarrhea* occasionally alternates with an extreme constipation.

The *general appearance* of the patient is termed chlorotic or "green," because it is a combination of a greenish-yellow color, with no marked tendency toward the loss of subcutaneous fat. The *sclera* present a pearl color or a bluish-white appearance, and this is often an early distinctive sign. There may be areas of pigmentation of the skin near the joints. Puffiness of the face and swelling of the ankles are indicative of nephritic complications. The facial color, however, is often deceptive, except in well defined cases, as the cheeks have a reddish tint, particularly after exercise. When the disease has existed for a long time pallor pervades the cheeks, lips and tongue, and even the fingertips. The *nervous disturbance* often becomes so exaggerated that hysterical symptoms are manifested by anesthesia of the skin, neuralgic crises, tinnitus aurium and the extreme mental perversions.

As the *circulatory disturbances* become more pronounced the cardiac palpitation increases so that the patient is not inclined to exercise. The pulsations of the carotids may be visible, the extremities are cold, and vertigo and syncope are not uncommon. *Soft systolic murmurs* are heard at the base or at the apex of the heart when there is an insufficiency of the mitral valve following left ventricular dilatation; a characteristic venous hum is detected over the jugular veins. The *urine* is generally increased in quantity, pale in color and deficient in the excretion of urea.

The *blood changes* contribute the most positive symptomatic indications. There is a decided reduction of the hemoglobin, with only a moderate reduction in the red blood corpuscles. The hemoglobin may fall to 40 or 38 per cent and in severe cases it may be much lower, while the red cells are not generally lower than 70 or 80 per cent. The white cells are decreased with a lymphocytosis. The red corpuscles are appreciably pale and moderately altered in size and shape. The majority are undersized (microcytes), while there may be a few which are abnormally large (macrocytes). Irregularity in shape—(poikilocytosis)—is often discovered, and it is possible to find a few small nucleated red corpuscles. A marked diminution in iron has also been determined.

Diagnosis.—The blood examination furnishes the most positive means of diagnosis, though it is essential to combine with this the typical symptoms. *Pulmonary tuberculosis,* with its pallor, feebleness and shortness of breath, is the most difficult to differentiate. This, however, should be differentiated by the physical signs, the greater emaciation, the tendency to night sweats, the cough and the peculiar temperature range. *Organic heart disease* will be disclosed by the valvular lesions and the signs of cardiac degeneration. *Nephritis,* which is often suggested by the pallor and edema, will be easily differentiated when the urinary examination is made. *Cancer* of the stomach, which is often simulated by the crises of epigastric pain, will be distinguished by the more constant anemia, the hematemesis, the paroxysms induced by the introduction of food and by careful physical examination and analysis of the stomach contents. *Cancer* and *pernicious anemia* will be recognized by a careful comparison in accordance with a symptomatic and pathological investigation in each case.

Prognosis.—If the disease is recognized in its early stages and the proper treatment is instituted, a favorable result may be expected in the average case. The majority of cases should improve decidedly in a few months' treatment.

Treatment.—*Hygienic regulation* is an important demand. *Outdoor life,* systematic exercise and a change of climate and surroundings are always to be encouraged. In some cases rest in bed will be required, while the rest cure treatment is always productive of much good. A *nutritious diet* should be given from the first and this should be rich in albuminous foods. *Digestive disturbances* should receive much attention both in the regulation of food and in the administration of the indicated remedies. Fats and carbohydrates are not to be allowed in excess, while stimulants like coffee, tea and wines should not be permitted. The *heart* may need special attention, but usually the indicated homeopathic remedies will suffice in this respect. The *nervous system* will be relieved of its perversions by the treatment mentioned under neurasthenia and hysteria.

Remedies have received much consideration in the management of this disease. In many cases they are not demanded and it should always be our aim not to load the system with drugs. *Iron* is regarded as the best medicine for internal use, but there is no doubt that its employment has been carried to an extreme degree. Many cases have, without doubt, been handicapped in their recovery by an overuse of this remedy. In severe cases, however, it is probable that a restoration of hemoglobin is gained by its use. Such preparations as *hemanutrine, ovo-ferrin* and *hemaboloids* may be administered after meals and are valuable adjuvants in this respect. A vigorous *nitrogenous diet,* however, may yield better results. *Blaud's pill,* in increasing doses, has been a favorite prescription and it is supposed to replace the iron which is reduced in the hemoglobin of the blood. The diluted tincture of the *chlorid of iron* has also many advocates. *Ferrum reductum* (grs. 2 to 3) three or four times daily, has also been highly lauded. The results obtained by the use of any form of iron will be appreciated by the counting of red corpuscles and the estimation of hemoglobin at regular intervals during its administration. *Quinin, strychnia* and *arsenic* have been used successfully in many preparations of iron.

In homeopathic doses we have used iron in the form of *ferrum phosphoricum* and *ferrum iodid* with decided success. *Arsenicum alb.,* and *arsenicum iodid* have probably been as efficacious as iron in the reduction of the anemia. Iron is called for when hemoglobin alone is low; arsenic is demanded when the count of red cells is markedly decreased. *China* is a close associate of arsenic in this respect and its combination, *china arsenicum,* is equally valuable. There are, however, other remedies which refer to the symptom totality of chlorosis and these should be given marked consideration; they are *calcarea carb., calcarea iodid, calcarea phos., calcarea fluorica, silicea, kali phos., sulphur, hepar sulphur, sepia* and *graphites.* Gastro-intestinal complications will call for remedies like *nux vomica, hydrochloric acid, bryonia, bismuth* and *carbo veg.* The general statement may be made that the patient should be treated in accordance with the suggestion of true symptoms as well as for the loss of hemoglobin. Under all circumstances we should place more stress upon dietetic and hygienic measures than upon the remedial agents.

PROGRESSIVE PERNICIOUS ANEMIA.

Progressive pernicious anemia, otherwise termed idiopathic anemia or Biermer's anemia, is characterized by a marked destruction of red blood corpuscles and a consequent decrease in their number, together with a reduction in hemoglobin which, however, is not proportionate to the corpuscular reduction.

Etiology.—The etiology is rather obscure, and it has been described by Addison as a "general anemia appearing without any discoverable cause." The researches of Quincke demonstrated a great increase in the iron in the liver and he therefore suggested that the disease might be due to an increased hemolysis. Hunter supported this view by the claim that the excreted urine contained a pathological urobilin. The hemolysis was explained as a condition following gastro-intestinal indigestion, in which infective agents are produced and, entering the blood, cause a destruction of the corpuscles. *Pregnancy* and parturition are looked upon as possible causes and *atrophy* of the *stomach* has been suspected, inasmuch as grave conditions of anemia have followed any atrophic change in the gastric mucous membrane. *Parasites,* such as the ankylostoma duodenalis and the bothriocephalus, have given rise to pronounced anemia and, therefore, are reckoned among the causes. Obscure *malignant diseases* and severe hemorrhages, diarrhea, fevers, mental shock and profound chlorosis are also regarded as having an etiological influence.

Bad *hygienic surroundings* and insufficient nourishment may favor it somewhat. *Sex,* apparently, makes no difference and no age is immune as it prevails during and after middle life although it has been observed in children.

Pathology.—Aside from the blood changes which are discussed under symptomatology, anemia is manifested in nearly every organ and tissue of the body, giving to the skin and tissues a pale lemon-yellow color. The muscles are an exception, being invariably red in color. The *subcutaneous fat* has the same lemon-yellow color, but is not generally reduced in amount, hence emaciation is not a pronounced feature. *Fatty degeneration* is one of the distinctive changes and it has a widespread distribution. The heart muscle shows pronounced fatty degeneration, the ventricles being more affected than the auricles. The *heart* is enlarged, flabby, friable and on section has a pale yellow color. Its cavities contain a small amount of blood, which is light colored. The *liver* also suffers from a similar fatty change, and is not increased very much in size. The walls of the gastro-intestinal tract, the kidneys and smaller blood vessels also show the fatty change. *Minute hemorrhages* are revealed in different parts of the body as the result of the degenerative condition. These may be observed in the retina and beneath the serous membranes. *Ecchymotic spots* may likewise be discovered on the skin and mucous membranes. *Edema* often exists in different parts of the body and accumulations of serum may be found in the pleurae pericardium and peritoneum. The lymph glands are occasionally swollen.

The *most characteristic change* is the increased percentage of iron in the outer and middle zones of the hepatic lobules. This deposit of iron pigment may also be detected in other organs like the spleen, kidney and pancreas. The *deposits in the liver* are no doubt due to the marked destruction of red corpuscles and are peculiarly distinctive of pernicious anemia. The changes in the *bone marrow* are marked, the yellow marrow being replaced by that which is deep red in color as the result of probable hyperplasia, which also may account for the increased number of large and small red cells and the presence of nucleated red corpuscles.

Lesions of the *central nervous system* are occasionally found postmortem, though the symptomatology does not suggest them during life. These for the most part partake of the general anemia, though small and at times large hemorrhagic extravasations are disclosed. *Hemorrhagic pachymeningitis* is a possibility and when the extravasation takes place in the cord a posterior and lateral sclerosis may follow.

Symptoms.—The symptoms are of such gradual onset that at first it will be impossible to diagnose the real disease. The patient usually complains of a feebleness which gradually develops into extreme exhaustion as the disease progresses. In exceptional instances they develop rapidly. *Pallor* is probably the first evidence, but as the anemia increases this is transformed into the typical lemon-yellow hue which pervades not alone the skin, but affects the mucous membranes also. *Headache,* vertigo and tinnitus aurium may be very annoying to the patient. *Dyspnea* and *cardiac palpitation* become distressing when the patient exerts himself. The anemia eventually becomes so profound that the slightest effort tends to produce syncope. *Gastro-intestinal* perversions are recognized by the anorexia, nausea and vomiting. Many of the symptoms incident to chlorosis are in evidence, but they are always more extreme. *Emaciation,* however, is not detected as the quantity of the body fat is preserved. *Edema* may be noticed about the ankles and the bodily torpor is soon supplanted by a pronounced mental hebetude.

Circulatory disturbances are prominent in this disease. Hemic murmurs, pulsations of the large arteries and veins and a soft, irregular pulse may be present, resembling that of aortic stenosis. *Pyrexia* has a pronounced significance as it is decidedly irregular in character. The temperature ranges between 101° and 102° F., and it is often as high as 104° F., when all of the other symptoms are usually aggravated. A subnormal temperature presages an unfavorable turn in the disease. The *respiration* is generally accelerated and in extreme conditions dyspnea is pronounced. When there are effusions into the pericardial or pleural sacs, pulmonary edema will be experienced. The *urine* has a low specific gravity, is dark in color on account of the urobilin, while the urea and occasionally the uric acid are increased in amount. The *nervous symptoms* are manifested by paresthesia, spastic rigidity of the muscles and the signs of graver involvement of the brain and spinal cord.

Hemorrhagic extravasations into the submucous, subcutaneous and subserous tissues are commonly observed. Retinal hemorrhage may give rise to amaurosis. *Epistaxis,* bleeding from the mouth and gums and metrorrhagia are frequent symptoms, and cerebral or spinal hemorrhages may be detected as the disease assumes the graver stages. The *bones,* particularly the sternum, are sensitive to pressure and the whites of the eyes, following the prolonged paleness of the conjunctivae, assume a peculiar pearly appearance.

The *blood shows* the most distinctive signs of the disease. In appearance it is usually pale, though it may, at times, be dark and watery. The most characteristic feature is the marked decrease in the number of the red blood corpuscles (oligocythemia), while the hemoglobin, although reduced in toto, is less so than the number of red corpuscles, thus giving to the red cells a high percentage of hemoglobin. It has been observed as low as 9 per cent. The number of red corpuscles is often reduced to one million, and records have been obtained which point to even a more decided reduction. Another peculiarity of the red corpuscles is the increase in their size (macrocytes) which may be detected in a majority of the cases. Other cells are smaller than normal (microcytes) while others are characterized by their irregularity in shape (poikilocytes). These changes, however, are not discovered in every case, so that they do not contribute a distinctive pathognomonic feature. Nucleated red corpuscles, however, are quite constant and these may be regarded as diagnostic of pernicious anemia, though they may appear in other diseases. They are discovered in two kinds, the normal-sized corpuscle, with its deeply stained nuclei (normoblasts) and a larger form with pale nuclei (megaloblasts). There are other signs of blood degeneration in pernicious anemia which may be detected in some instances. There is generally a relative increase in the small lymphocytes, while a marked leukopenia has been discovered in some cases. Myelocytes are occasionally present, but are not numerous. Michrost's corpuscles, highly colored spherical bodies, have also been found. The blood plaques are either absent or very scanty.

Diagnosis.—When a painstaking study of the symptomatology is undertaken confusion in diagnosis ought not to exist. The *profuse anemia,* with its lemon-yellow tint, the progressive and extreme exhaustion, the *digestive* and *cardio-vascular* symptoms, the *reduction* of the red blood corpusles to one million or less in number, the proportionate diminution of hemoglobin with an occasional relative increase, the presence of macrocytes, microcytes and poikilocytes, together with the red nucleated corpuscles and other minor blood changes, should constitute the distinctive symptoms for diagnosis. The fever, the hemorrhagic tendency and the obstinacy to treatment are also characteristic symptoms. There should be no confusion in the case of *chlorosis,* which almost invariably occurs in young women during adolescence, while the blood changes are distinguished by a reduction of hemoglobin out of proportion to the decrease in the number of red corpuscles. *Secondary anemia* will be identified by the discovery of some organic condition which accounts for it. *Addison's disease* will be recognized by the skin pigmentation, a milder anemia and the absence of the typical blood picture observed in pernicious anemia. *Tuberculosis* should offer no difficulties in differentiation when the comparative symptomatology is considered, and the same may be said of *renal* and other diseases attended by anemia. *Intestinal parasites* will not be confounded when the parasitic evidence is found, and *atrophic gastritis* will be distinguished when the gastric contents are examined. In *gastric cancer* the red corpuscles are not reduced so much, or at least the reduction does not keep pace with the extreme cachexia and emaciation; the disease occurs after middle life and the gastric symptoms should help to differentiate. *Leukemia* should offer no diagnostic perplexity when this disease with its peculiar blood picture is carefully considered.

Prognosis.—A fatal termination may usually be expected, though there may be favorable remissions of the severe symptoms at times. While the disease pursues a slow course there is nevertheless a gradual and pronounced loss of strength until life fades away. Typical cases often terminate in death in a few months, or at least in a year. Death may result from asthenia, syncope or cerebral hemorrhage.

Treatment.—In the treatment of a disease so hopeless as pernicious anemia we can not expect much from our remedies and the general care. In order, however, to palliate and prolong life we must resort to every means to that end. Hygienic care and nutritious food will always be worthy considerations. *Arsenic* has been considered as a remedy which might delay the destruction of corpuscles to a certain extent, and Fowler's solution has been regarded with unusual favor. This has been prescribed in increasing doses until twenty to thirty minims have been given three times daily. *Arsenious acid* in pill form (gr. 1-50 to 1-30) has also been recommended three times daily. It has also been employed in conjunction with iron and strychnia. *Sodium carbonate* may be used hypodermatically. *Bone marrow* has received some praise as a remedy in this disease, but sufficient data is not at hand to warrant much commendation. *Antistreptococcus serum, intestinal antiseptics, saline solutions* and many other measures have been employed with the possible hope of obtaining temporary if not permanent relief.

Consideration from the homeopathic standpoint is equally discouraging, though it is essential at all times to regard the symptoms in prescribing for intercurrent conditions. Life may possibly be prolonged when we relieve the complicating features. *Arsenic* must be regarded as a prominent and useful remedy the same as when employed in heroic doses. Goodno also suggests the use of *nitric acid* and the *picrate of zinc.* It is probable that special symptoms may call for *cinchona, chinium sulph., acetic acid, sulphur, plumbum, phosphorus* and other indicated remedies.

LEUKEMIA.

This disease is also known as leukocythemia, and is characterized by an increase in the number of leukocytes and also by changes in the bone marrow, spleen and lymphatic glands. It is considered as acute or chronic according to the rapidity of its development.

Etiology.—We have no definite knowledge as to its cause. It is observed in all countries and shows no special predilection for sex or age. The majority of cases properly develop during middle and later life, though I once observed a case in a young girl sixteen years of age. *Heredity* has been sought as a cause, but we have no positive confirmation of this. *Malaria,* syphilis and pregnancy are said to have produced the disease and have been considered as etological factors, as have nerve strain, traumatism and unhygienic conditions. The probability of *infectious origin* has received considerable attention, but no definite micro-organism has been discovered as a cause. Its frequent association with autointoxication has given some excuse for this theory, though a direct relation can not be clearly established. Intestinal ulceration and such diseases as stomatitis should be studied in this relation, as should typhoid, pneumonia, variola and rheumatic fever.

Pathology.—The pathology of leukemia is clearly marked by changes in the blood, the spleen, the lymphatic glands and the bone-marrow. These alterations are recognized under two principal groups which typify the pathologic process: (1) The *spleno-medullary* (or splenic-myelogenous) leukemia, which is the most common form, and (2) *lymphatic leukemia,* The general features of each of these will be recognized by the *extreme emaciation* and *pallor,* with occasional edema and dropsical effusions. The heart and veins are distended with large greenish-yellow blood clots which, on account of the increase in leukocytes, may assume a pus-like appearance. The pericardium and endocardium may show ecchymoses while the myocardium often undergoes a slight degree of fatty degeneration. The *blood* may contain certain albuminous substances and also leucin, tyrosin, acetic, formic and lactic acids. The alkalinity of the blood is usually reduced, the fibrin is increased, the specific gravity is lowered and Charcot's octohedral crystals may be discovered in it.

In the *spleno-medullary* form we shall find the *spleen greatly enlarged.* It may be adherent to the abdominal walls, diaphragm, stomach and other viscera because of a perisplenitis, while the capsule is generally thickened and the vessels at the hilus are enlarged. In the first stage of splenic involvement the organ is hyperemic, soft and swollen, the Malpighian bodies being only slightly hyperemic and enlarged in comparison to the swollen pulp. In the second stage the Malpighian bodies show the effects of the hyperplastic changes and as they increase in size they may encroach upon the pulp. The third stage is characterized by the granite-like appearance of the spleen, in which grayish white lymphoid bodies are interspersed throughout the organ, contrasting strongly with the reddish-brown matrix. The spleen in this stage is hard and cuts with resistance. Its size is enormous and it may weigh as much as eighteen or twenty pounds; it often fills the greater part of the abdominal cavity on the left side, and projects well down toward the pubes, the enlargement being uniform, while the exaggerated notches of the anterior border are quite distinctive.

The *bone marrow* also suffers a decided structural change; the fatty medullary substance is transformed into a mass of lymph cells with nucleated red corpuscles in all stages of degeneration. The marrow is either reddish-brown or greenish-yellow in color, the transformations being termed respectively "lymph-adenoid" and "pyoid." These two forms of degeneration may be seen in alternation or a fine reticulum is often interspersed between the cells, while small hemorrhagic infarcts are sometimes present. Nucleated red corpuscles in various degrees of development may be observed. Cells resembling the myelocytes may also be found. The *lymphatic glands* are slightly enlarged, but not to such an extent as in lymphatic leukemia.

The *lymphatic variety* is characterized by a general lymphatic gland increase and with this there is a slight enlargement of the spleen. The cervical, axillary, mesenteric and inguinal glands are chiefly affected, being enlarged, soft and movable, having a reddish-gray color. The tonsils, the lymph follicles of the tongue, pharynx and mouth may be involved, as also may the thymus gland and the solitary and Peyer's patches. This pathology is distinguished by a true hyperplasia.

The *liver* is also decidedly enlarged, cases being quoted in which it weighed as much as fourteen pounds. The enlargement is the result of leukemic infiltration and the growth is uniform. The capillaries and interlobular tissue are filled with leukocytes, while lymphoid cells undergoing cell-division are discovered in considerable abundance. Definite leukemic growths are also frequently found. The kidneys are occasionally similarly affected, being enlarged and pale in color. Leukemic tumors may be found also in the brain, lungs, retina, serous membranes, testicles and skin.

Symptoms.—*Acute leukemia* has been defined as a rare form in which the pathologic features develop with great rapidity and death occurs in one to two months' time. *High fever,* hemorrhages from mucous membranes and profound prostration characterize the onset. The *splenic enlargement* and sometimes the lymphatic growths develop with astonishing rapidity. *Cardiac excitement* should be suspected from the pronounced dyspnea. *Gastro-intestinal disturbances* are very severe. *Anemia* is marked and edema is common. The leukocytes rapidly increase, often having a ratio of one to thirty with the red corpuscles; the lymphocytes are always extremely numerous, the same being true of the mononuclear leukocytes and myelocytes; the eosinophiles are few in comparison to the blood condition in the chronic form. As the case progresses, hematemesis, cerebral or retinal hemorrhages and purpuric manifestations may be evidenced.

Chronic leukemia will show a more gradual onset and the general symptoms are often so insidious in their development that they are not usually recognized. The patient may complain for a long time of a *distressing languor* which can not be accounted for. *Pallor* is indicative of the increasing anemia; pronounced dyspnea exists upon exertion and such symptoms as *headache, exhaustion, faintness* and *digestive disturbances* will evidence the progress of a severe exhausting disease. A *moderate fever,* often reaching as high as 103° F. may be noticed later, while *hemorrhagic manifestations,* in the form of epistaxis, hematemesis, enterorrhage and retinal hemorrhage are of occasional occurrence, and *hemic murmurs* are often detected. The *pulse rate* is increased and the quality may alternate between a soft and compressible pulse and one which is full in volume. Edema may be a comparatively early symptom, while *ascites* and *jaundice* may appear as later developments. *Dysenteric diarrhea* is an indication of the ulcerative process in the intestines and *leukemic peritonitis* is an evidence of lymphomatous growths in the peritoneal membrane. *Nervous symptoms* may be so pronounced that syncope ensues and sudden coma and hemiplegia may follow an unexpected apoplectic attack. The *urine* may contain an abundance of uric acid and in extreme states albuminuria may be present.

The *spleen* gradually increases in size until it fills a large part of the abdominal cavity. This may be attended by some pain in the splenic region or it may develop without any sign of tenderness or pain. The normal outline of the organ will usually be detected by careful palpation, and it may display a sudden increase in size after a severe hemorrhage or diarrheal attack. Constipation may also attend the splenic and hepatic increase. Pulsations and a "splenic souffle" or systolic murmur may be discovered over the tumor. The lymphatic enlargements will be found in the splenic-lymphatic variety and these are invariably soft and movable. Tenderness over the sternum and long bones may sometimes be detected.

The *blood changes* are most characteristic of all the symptoms. In the spleno-medullary form the blood is pale in appearance and often assumes a brownish-red or chocolate color. The *leukocytosis* is the most pronounced feature. The ratio of the white to the red cells may vary from 1 to 150 down to 1 to 10, or 1 to 5. The *myelocytes*—large mononuclear leukocytes, with the protoplasm filled with neutrophilic granules—may form one-fourth of the white cells; these are not found in normal blood. The polymorphonuclear cells are generally relatively diminished below their percentage in normal blood, while the same cells, which show coarse basophilic granules, are increased. The lymphocytes are relatively decreased, while the acid-stained eosinophiles usually show an absolute increase. The total number of blood cells, including both the white and colored are decidedly reduced, being sometimes as low as 2,000,000 and 3,000,000. Suchannek mentions a case in which the red cells were only 301,600 and the colorless corpuscles were 306,100. *Nucleated red discs* are usually discovered and poikilocytosis is not uncommon. The *hemoglobin* falls below the normal and the blood plaques may be slightly increased. The blood coagulates slowly and the alkalinity may be somewhat diminished. The specific gravity averages 1030 to 1050. In *lymphatic leukemia,* which is more fatal, but fortunately rarer, the lymphocytes are increased while other leukocytes are relatively diminished.

Diagnosis.—The only trustworthy diagnosis is obtained by a thorough blood examination. *Splenic anemia* will be frequently taken for this disease, the anemia being fully as great, while the spleen is often greatly enlarged. In this, however, the red cells are near the normal count, though the hemoglobin may be reduced to 50 per cent or lower. Poikilocytosis may exist and leukopenia is generally recognized, but myelocytes are never found in the blood. *Chronic malaria* may simulate leukemia as the spleen enlarges. In this, however, the chills, the history of acute malaria, the absence of lymphatic enlargement, with only a slight increase in the white cells, will aid the differentiation. *Hodgkins' disease* will be distinguished by the marked enlargement of the cervical lymphatics in bunches, while the blood shows only a leukocytosis.

Prognosis.—The prognosis in this disease is always unfavorable. Death usually occurs in two to five years, while the more rapid cases may prove fatal in six to eight months. Temporary improvement may take place in some instances when the patient's vitality is maintained by food and remedies. An unfavorable exacerbation is, however, liable to take place unexpectedly, when the disease displays a more progressive and pronounced symptomatology. Profuse diarrhea, cerebral apoplexy, hemorrhages of various kinds, together with severe exhaustion will often precede the sudden demise. Any intercurrent disease will hasten death.

Treatment.—Thus far nothing has acted curatively in this disease. To overcome the extreme exhaustion nutritious food and outdoor life are essential. Remedies may help somewhat in this respect, but they will insure no permanent relief. *Arsenic* has been praised for its apparent power of retarding the severe blood changes. That something is obtained in this respect we can not deny, but the disease will soon get the better of any remedy. *Quinin, iron, bone marrow* and the usual expedients thought of in simple anemia are often tried, but they are at best only temporary measures. When a patient is afflicted with this disease death is the one termination to be expected. Our aim should be to relieve the symptoms of intercurrent affections and at all times to sustain the patient's strength as much as possible. Among the remedies which are usually indicated we shall find *picric acid, thuja, phosphorus, strychnia, nux vomica, cinchona, natrum sulph.* and *sulphur* of some use from the standpoint of symptomatology.

Recently the use of the X-ray has been highly commended, but more extensive reports are desired.

PSEUDO-LEUKEMIA

Hodgkins' disease is the usual term employed to define this disease; it is also described under other terms such as lymphatic anemia, lymphatic adenoma, malignant lymphoma, etc. It is characterized by a progressive enlargement of the lymph glands with secondary lymphoid growths in other organs, particularly the spleen and liver, and by a marked secondary anemia without the destructive blood changes which are found in true leukemia.

Etiology.—The true nature of the disease is undetermined. *Age* is a predisposing factor, the majority of cases appearing between twenty and forty years. *Heredity,* syphilis, malaria and tuberculosis have been regarded as favoring influences, but nothing definite can be said in this respect. Some infectious agent, especially the tubercle bacillus, has been thought of as an exciting cause. This would seem to be a plausible theory, but as yet there has been no confirmation of this claim by bacteriological discovery.

Pathology—The morbid anatomy has a distinctive feature in the *enlargement of the lymphatic glands.* This change will be observed first in the more superficial glands of the anterior and posterior cervical triangle, while the axillary, inguinal and retroperitoneal glands may soon be implicated. In fact, all the glands of the lymphatic system are subject to this hyperplasia, including the scapular, pectoral, mediastinal and bronchial, the enlargements showing a particular affinity for the glands distributed along the large blood vessels. The glands, singly or in groups, may become quite large and, at first, may be freely movable, while later they may become more or less fixed by a periadenitis. This enlargement of the glands may cause pressure-symptoms, especially those situated near the large vessels and important nerve trunks. On section they present a white or reddish-gray appearance, but as they become firm they assume a yellow-white color. Hyperplasia of the glandular elements characterizes the pathology. Secondary changes similar to caseation and fibroid induration may be observed, while suppuration takes place only as the result of prolonged local irritation and the introduction of pyogenic infection.

The *spleen* suffers only slight enlargement from this hyperplastic development, both the pulp and the Malpighian bodies being affected. The change in the latter is a real hypertrophy, giving them a grayish-white color which contrasts vividly with the normal dark-red color. When the pulp is affected it shows a trabecular overgrowth. In some cases the spleen is not enlarged. The lymphoid deposits may also be found in the liver, kidney, lungs, thymus gland, brain, tonsils, intestinal lymphatics and nerve tissues. The bone marrow also may suffer a similar lymphoid change.

Symptoms.—*Enlargement* of the glands on one side of the neck is the first symptom which calls attention to the disease. These undergo a gradual increase in size, causing a conspicuous disfigurement. After a time the glands on the opposite side of the neck begin to enlarge, and in the course of a few months, or perhaps a year or more, the axillary glands are affected and then the inguinal and internal lymph glands may show signs of enlargement. *Pain* may be marked when there is pressure on nerve trunks. A chronic tonsillitis often precedes the glandular increase.

Constitutional symptoms will be evidenced by the pallor of anemia and exhaustion which appear about the time the cervical enlargement is detected. Emaciation is soon noticed, while gastro-intestinal disturbances, headache, dizziness, cardiac palpitation and dyspnea appear as the disease progresses. *Hemorrhage* in the form of epistaxis, metrorrhagia, and petechial spots on the limbs are occasionally observed and effusions into the serous cavities are not uncommon. *Hemic murmurs* may be detected, while *fever*, often of an intermittent type, is common in this disease.

Mechanical pressure will give rise to pain in different parts of the body whenever a tumor presses upon a nerve. In like manner the enlargement of the tracheal and bronchial glands may cause dyspnea as they press upon the trachea or bronchi, or dysphagia when the esophagus is compressed, or disturbed phonation when the recurrent laryngeal nerves are affected, or venous distention when the superior vena cava or jugular veins are encroached upon. Pressure upon the pneumogastric nerve will disturb the heart's action, while the enlargement of mediastinal glands may displace the heart itself. *Edema* is liable to follow from venous compression. *Ascites, albuminuria* and *jaundice* are possible symptoms from the same cause. Pressure upon the cervical sympathetics may cause inequality of pupils and unilateral sweating of the face and neck. *Paraplegia* is also a possibility when the spinal cord is involved. *Bronzing of the skin* is a rare symptom and is believed to be due to pressure upon the adrenals. *Pruritus* is also a common skin affection. The *spleen* is moderately enlarged while tenderness and nodular enlargements may be occasionally detected by palpation; the splenic enlargement, however, is much less than that of leukemia.

The *blood changes* are a feature of the disease, but in no sense are they so marked as in the other diseases of the blood which this simulates. The color is somewhat pale and there is a moderate diminution in the number of red blood corpuscles and a corresponding decrease in hemoglobin, which is reduced, on the average, to 50 or 60 per cent. An occasional nucleated red blood corpuscle and a poikilocyte may be found. The leukocytes may be increased, but this is a rare finding. In no sense do the blood changes approximate those of leukemia.

Diagnosis.—*Tuberculous adenitis* is most frequently confused with the pseudo-leukemia in its earliest stages. Tuberculous glands, however, become adherent earlier and suppuration is more liable to take place, while caseation is quite common; they enlarge more slowly, are inclined to be unilateral and usually only single groups are involved; the young are most commonly affected, while pseudo-leukemia may occur at any age. Gowers observed in 100 cases that only one-third occurred under twenty years of age. In tuberculous adenitis lesions of the lung may be found and the other typical symptoms of tuberculosis are liable to develop; the pyrexia is nocturnal, while in pseudo-leukemia it is characterized by intermittency. *Sarcoma* involves the glands in groups, rapidly invades surrounding tissue and is early characterized by a destructive ulceration. *Carcinoma* of the lymphatic glands resembles the growths in pseudo-leukemia, but the cancer cachexia is distinctive and the glandular invasion is secondary. In all of these diseases which are compared with pseudo-leukemia, the gland involvement is usually limited to single groups, which is not true of this disease. When a careful blood analysis is made, there should be no difficulty in differentiating leukemia except possibly in the acute lymphatic form. Sections of the involved glands should be examined microscopically to remove any doubt.

Prognosis.—A fatal end is the usual termination of this disease. Patients affected rarely survive longer than two years. The fatal symptoms are hemorrhage, anasarca, and progressive asthenia, while a rapid increase in the size of the glands and a continuous fever usually presages a speedy termination.

Treatment.—Inasmuch as the disease is inevitably fatal, it seems futile to say much regarding remedies. Still there is a fair possibility of palliation and the prolonging of life. I have often felt that my treatment had comforted the patient decidedly and in some instances added to the length of life. *Arsenic* has been heralded as the only remedy and this is given in gradually increasing doses. Fowler's solution is still advised as the best form for use. It has been employed hypodermatically by some, eight to thirty minims being used in divided doses daily. *Inunctions* of iodin and iodid of potash have also been suggested, as has the employment of quinin, cod liver oil, bone marrow and many other internal agents which would naturally support the system against the depletion of anemia. All of these expedients, however, are only temporary and hardly that unless they are applied during the early stages of the disease.

In my experience with several incipient cases, I have found that a stimulating and vigorous diet, with particular reference to the restoration of hemoglobin, together with the indicated homeopathic remedy, has given me some encouragement. In the list of remedies I would call special attention to *calcarea iodid, arsenicum iodid, ferrum iodid, kali iodid, iodin, iodoform, phosphorus, sepia, silicea* and *sulphur.* A cure is not expected from their use, but they may at least relieve the patient and delay somewhat the onward march of the disease.

Local treatment of the glandular enlargements has not availed much, though all kinds of measures have been tried from galvano-puncture to iodin, blisters and massage. *Operations* may be demanded for relief of pressure, but up to the present date surgery has been of no avail. So far we may safely state that the disease is incurable. The use of the X-ray has given some encouragement in treatment, though sufficient data are not yet obtainable upon which to base positive conclusions.

STATUS LYMPHATICUS.

This is also termed "lymphatic constitution" and applies to a state characterized by *hyperplasia* of the lymph nodes, spleen and thymus gland, and *hypoplasia* of the heart and arteries. The lymphoid bone-marrow is also involved in this condition. The disease is met most frequently in children or infants and sudden death is liable to take place when the child is apparently in robust health.

Etiology.—While the etiology is not fully understood it is presumed to be a congenital condition associated with a low grade of vital resistance. Blumer claims it is due to the development within the system of a toxic agent known as cytotoxin, which causes the overgrowth of lymphatic tissue.

Pathology.—The lymph glands in the thorax and abdomen and the cervical and inguinal glands are enlarged. Those of the intestine are swollen and sharply defined. The tonsils are enlarged, the spleen is decidedly increased in size and the lymphoid tissue of the Malpighian bodies also manifests the hyperplasia. The thymus gland is decidedly swollen and it is claimed that the pressure caused by this pathologic increase often causes sudden death. The thyroid gland may also be enlarged. The red bone marrow is supplanted by yellow marrow, the lungs are congested or edematous, while the heart and aorta are not fully developed.

Symptoms.—While these are not clearly defined symptoms it will be observed that the child has an enlargement of some of the lymphatics; there may be a history of rickets and the heart is weak in its action. From overexertion or the administration of an anesthetic such children are liable to sudden and unexpected death. Tetany has been recognized as a complication by Hand. In some instances deaths attributed to convulsions during dentition were really due to this disease.

Prognosis and Treatment.—From the foregoing it is evident that it is rarely possible to make a diagnosis of this disease. For this reason there is little to be expected from treatment. The general health should be supported by outdoor life, nourishing food and possibly aided by such remedies as *iron* and *arsenic*. Operation upon the thymus gland has been successfully performed. We should recognize that the overplump children with enlarged glands are liable to sudden development of grave symptoms and should, as far as possible, increase their vital resistance. Such remedies as *calcarea carb.*, *calcarea phos.*, *sulphur*, *arsenicum iodid*, *silicea*, *baryta carb.*, and *baryta iodid* should be studied in these cases.

SPLENIC ANEMIA.

There is some doubt still about the correctness of the classification which considers this disease as a distinct variety of anemia. It is analogous to pseudo-leukemia, yet distinguished from it by the absence of lymphatic enlargement. In all probability it is closely associated with infectious diseases, though the etiology is not yet clearly understood. Chronic malaria has been considered as a possible cause, but a careful study of this disease does not establish any definite relation in this respect.

Pathology.—The typical morbid feature is the splenic enlargement which simulates more closely the leukemic spleen than the spleen of pseudo-leukemia, inasmuch as it is often three or four times its normal size. The normal shape is retained, though the notches may be decidedly accentuated. The capsule is thickened and often adherent to surrounding tissues. The histologic findings exhibit splenic atrophy with an attending sclerosis of the Malpighian corpuscles, which is, of course, preceded by a fibrosis or hyperplasia. Changes in the bone marrow are sometimes discovered similar to those found in pernicious anemia.

Symptoms.—The *early symptoms* are marked by the signs of pallor, exhaustion, dyspnea, palpitation and the distinct evidence of a developing anemia and splenic enlargement. The *second stage* is characterized by a more profound anemia, extreme emaciation, with physical exhaustion, pain in the region of the spleen due to enlargement, hemorrhages from mucous membranes and even of the skin. Added to this there is usually pronounced hebetude. The *final stage* represents a progressive asthenia which ends in death. Jaundice and ascites may be present should the liver become involved.

The *blood characteristics* are indicative of anemia. The hemoglobin is greatly reduced—often as low as 40 per cent—while the number of the red cells may be diminished at least one-half. Poikilocytosis often exists and there may be numerous nucleated red-blood cells. The leukocytes are usually increased, though in some cases they may be normal, or nearly so.

Diagnosis and Prognosis.—The diagnosis will depend upon the splenic enlargement plus the symptoms of marked anemia, just enumerated, together with the absence of lymphatic enlargement. The blood picture should also be confirmed. The prognosis is unfavorable, though by proper treatment life may be prolonged in many instances. The average duration is from six months to two years.

Treatment.—The treatment should be similar to that suggested under other forms of anemia. *Iron* and *arsenic* are regarded as the best remedies, though many other remedies may be suggested by the totality of the symptoms. *Bone marrow* is, without doubt, a valuable adjuvant. In a case of mine this, together with the constant use of *arsenicum* and a judicious diet in which meat juices were given in abundance, contributed toward a remarkable improvement which prolonged the patient's life for a considerable time. In this case the red cells were reduced to nearly two million, while the hemoglobin stood at 45 per cent. After a persistent treatment the hemoglobin was increased to 70 per cent, but no increase was obtained in the number of red cells. Splenectomy has been advised when hematemesis is persistent, and has proven successful in a few cases.

Banti's Disease.—This term is used to define a condition characterized by enlargement of the spleen, by anemia, hepatic cirrhosis, jaundice and ascites. It is a very rare disease and is regarded by many as a terminal stage of splenic anemia. The evidence, however, goes to prove that it is sometimes a primary, though very rare, disease.

ANEMIA INFANTUM PSEUDO-LEUKEMIA.

This is a rare form of anemia first described by von Jaksch and observed in children two or three years of age. It resembles leukemia without its fatal termination. Hereditary syphilis, intestinal and other diseases are supposed to be factors in its development. The typical lesion is a splenic enlargement following a uniform hyperplasia. The liver may also be enlarged and the lymphatic glands may be slightly increased in size, though there is no apparent tumor-formation. Some changes may be found in the bone-marrow.

Symptoms.—Associated with a gradually developing pallor, exhaustion and emaciation there will be noticed an enlargement of the spleen, which may attain a large size. The liver is slightly enlarged and gastro-intestinal disturbances are commonly experienced. Emaciation ensues if the disease is of long duration. The *blood changes* reveal a decided decrease in the number of red corpuscles, while there is generally a relatively greater reduction in the hemoglobin. Poikilocytosis, a large number of nucleated red corpuscles and other signs of degeneration in the red cells may appear in the graver forms. There is always a marked increase in the number of leukocytes.

Diagnosis and Prognosis.—While the classification of this disease will depend upon the distinctive symptoms just mentioned it will be necessary to differentiate from leukemia. This is settled by the absence of hemorrhage and lymphomatous enlargements and the presence of an abundance of nucleated red corpuscles, gastro-intestinal symptoms and the signs of rachitis. The leukocytes will also constitute a distinct differentiation from leukemia. The prognosis is comparatively favorable.

Treatment.—Dietetic and hygienic treatment are of paramount importance. The digestive disturbances will also be corrected by the employment of the usual homeopathic remedies. The anemia will be cared for as suggested under other chapters.

CHLOROMA.

This is a rare disease which has a slight resemblance to leukemia and pseudo-leukemia. It is characterized by a sarcomatous growth with a pea-green pigmentation affecting the periosteum and bone tissue in the vicinity of the orbit. Metastatic nodules representing secondary growths have a widespread distribution. The disease is prone to appear with greatest frequency in young adults.

The symptoms are not numerous; orbital pain, exophthalmos, deafness, conjunctival hemorrhage and epistaxis are the most distinctive signs. The growths are round and elastic, being found most frequently in the orbital, temporal and parotid regions. The blood shows an anemia with a marked leukocytosis. The disease has been termed the "green cancer." It is rapid in its development and death usually occurs in a few months.

DISEASES OF THE DUCTLESS GLANDS.

DISEASES OF THE SUPRARENAL CAPSULE.

ADDISON'S DISEASE.

This is a constitutional disease characterized by a bronzed or pigmented skin, general asthenia, cardiac debility and gastro-intestinal disturbances. It is believed to depend upon degeneration of the adrenals or changes in the sympathetic semilunar ganglia, or to both.

Etiology.—This is very indefinite. The disease is observed usually between 20 and 50 years of age, though it has been discovered in young children, and may even be congenital. Males are afflicted more frequently than females. Overwork, nervous exhaustion and injury of the back or abdomen have been considered as causes. Infection, particularly the tuberculous, is probably a factor of great influence in its production.

Pathology.—The adrenals show decided changes in a great majority of cases. These changes are usually tuberculous, though much less frequently are one or more of the following conditions observed: Simple atrophy, chronic interstitial inflammation, malignant disease and hemorrhagic extravasations into the suprarenal bodies. Pressure on, or inflammation of the semilunar ganglia is also held to be a cause of this disease. Whether the involvement of the semilunar ganglia is primary cr secondary to changes in the adrenals is a matter open to much discussion. Probably, however, the real cause lies primarily in the adrenals, and is due to suppression or perversion of their internal secretion. Other pathologic changes include enlargement of the solitary and agminated follicles of the intestine, enlargement of the spleen, atrophy and fatty degeneration of the heart, kidneys and thymus gland.

Symptoms.—In studying the symptomatology of Addison's disease we must keep in mind the fact that the symptoms are often so insidious in their development that they do not present a perfect picture of the disease. *Pigmentation,* or bronzing of the skin, is the most typical sign; but must be accompanied by other symptoms of this disease. The cutaneous discoloration varies from a light yellow to a deep brown and in some cases it may assume a black color. The deeper hue is always observed on exposed parts and where the normal pigmentation is marked, like the face, neck, hands, abdomen, groin, genitals and the areola of the nipples. These spots are occasionally found on the mucous membrane of the mouth, lips, conjunctiva and vagina, when they are liable to assume a bluish color. *Leukoderma patches* are frequently associated with the bronzing, causing thereby a marked contrast with their absence of pigment. In rare instances there may be a general bronzing. The typical patches usually shade into the normal skin, thus presenting no well defined line of demarcation.

Some have claimed that the pigmentary deposit may be found in the internal organs, but no satisfactory demonstration of this theory is at present on record.

Constitutional symptoms are generally recognized as the disease develops and sometimes they may precede the appearance of the bronze-colored patches. *Extreme debility* is a constant complaint and with it we shall detect such characteristic symptoms as headache, dizziness, ringing in the ears and breathlessness, with a gradually progressing asthenia which leads to a marked loss in physical and mental energy. *Anemia* is not pronounced. The blood examination shows only a moderate reduction of red blood corpuscles and hemoglobin. A leukocytosis is rarely found, a leukopenia usually taking its place. The heart's action is feeble and the pulse is small and rapid; thus dyspnea and a tendency to faint are frequent complaints. Functional cardiac murmurs are not uncommon and the extremities are often cold and clammy. *Gastro-intestinal* disturbances are prominent features in the symptomatology and they will be recognized by the loss of appetite, nausea and vomiting, which occur without any apparent cause, and a debilitating diarrhea, which frequently exists during the later stages. Hebetude, merging into coma, may appear in the later stages of the disease while progressive asthenia is usually the cause of death. *Polyuria* is often a symptom, but albumin is rarely discovered; indican is, however, increased, urea is decreased, while urobilin and uromelanin may appear in excess.

Diagnosis.—The diagnosis should comprehend all the symptoms and never be made on the presentation of the typical bronze patches alone, since these may appear in association with other diseased conditions. We may find a similar discoloration in *tuberculosis of the peritoneum* or in *cancer* when located in the abdomen. It is also found in *hepatic disease* and especially in *diabetic cirrhosis*. *Pregnancy* and uterine diseases often give rise to similar patches on the face. The discoloration of vagabond's disease may simulate that of Addison's disease. *Melanotic sarcoma* is accompanied by a deep general pigmentation. We may also find the simulating bronze spots in such diseases as tinea versicolor, exophthalmic goitre, old syphilitic discolorations and from the use of such drugs as silver nitrate locally and the prolonged internal use of arsenic.

Prognosis.—The disease is almost always fatal, though there may be periods of improvement which offer the hope of a cure. It pursues usually a rapid course after the characteristic symptoms appear. One year is given as the average duration for the disease, though in exceptional cases it may develop rapidly with febrile symptoms and terminate in sudden death. The end usually comes as a result of the progressive asthenia, but coma, delirium or convulsions may characterize the termination.

Treatment..—In a disease so fatal as this we should not expect more than palliation from the use of our remedies. This, however, may be an important consideration in the prolongation of life and comfort. *Hygienic care* and rest are essentials always to be thought of and we may do much toward the delay of the asthenic advance by the employment of these measures. The *digestive disturbances* will give at least temporary response to the indicated remedies and we may do much toward heart stimulation by the use of the tonics usually prescribed for this purpose. *Iron* and *arsenic* have been lauded as useful remedies inasmuch as they tend to correct the anemia and the incident prostration. Whether they yield any beneficial result, so far as the disease is concerned, can not yet be stated. *Adrenalin* is naturally thought of. Osler, Shattuck, Deeks, Porpelow and others have reported cases cured and others greatly improved by its administration. It should be given cautiously and in small doses. In the later stages and severe forms of this disease it is contraindicated.

Among the homeopathic remedies we may find several which conform to the symptomatology, but these apparently serve only to correct the attending symptoms of associate perversions. *Arsenicum iodid, baryta iodid* and *calcarea iodid* should receive especial emphasis. *Argentum nitricum* and *theridion* have received special mention as remedies presumably indicated for the symptoms which express the pathologic changes. It is probable that others may be called for in this respect. *Phosphorus, guaiacol* and *iodoform* would be naturally considered when there are manifest signs of tuberculous invasion of the lungs and other tissues. It would be natural also to give due weight to the open air treatment for tuberculosis. The *nuclein* preparations have also been suggested.

DISEASES OF THE THYROID GLAND.

THYROIDITIS.

This is an acute inflammatory condition of the thyroid gland with or without any association of previous enlargement. The gland is swollen, the blood vessels are engorged, while hemorrhage, thrombi, multiple abscesses and tissue necrosis are commonly found. The inflammatory invasion is invariably secondary to the introduction of an infectious micro-organism; it is, therefore, liable to follow in the course of such diseases as malaria, typhoid fever, variola and, possibly, rheumatism. Traumatism is a possible cause, inasmuch as this may establish a strumitis which favors the invasion by pyogenic organisms either from within or without the body.

The *symptoms* are characteristic of suppurative development, hence, fever, localized pain and swelling are early signs. When the abscesses are multiple the whole gland may become very large, but usually the enlargement is confined to one lobe. The symptoms of venous obstruction may be marked, while compression upon the trachea may interfere decidedly with respiration. The pus may burrow into surrounding tissues or the abscess may perforate the trachea or esophagus. Metastatic development is always to be feared, though resolution may take place without any prolonged or serious complications. The prognosis is favorable when by rupture or operation the pus is evacuated. The treatment will demand prompt and careful surgical interference, together with the internal use of remedies which are applicable in suppurative cases. *Hepar sulphur, arsenic, mercurius, iodoform* and *silicea* should be considered.

GOITRE.

A simple goitre has been described under various terms, such as bronchocele, thyrocele, Derbyshire neck, etc. It is defined as an enlargement of the whole or a part of the thyroid gland other than that due to inflammation, exophthalmic goitre and malignant disease. It must be clearly differentiated from exophthalmic goitre, which is an enlargement of the thyroid gland as the result of a nervous disease.

Etiology.—In America goitre is of sporadic occurrence, but it has appeared endemically in Italy, Switzerland. Asia and in certain parts of England. Some localities in those countries are noted for the abundance of goitrous inhabitants, in some instances as many as eighty per cent of the population being afflicted. It has a wide prevalence in localities where water containing much lime has been constantly used. It has been noticed that people who move into a locality where it prevails develop the enlargement, thus furnishing a satisfactory proof that it may be traced to the water supply. *Women* are evidently afflicted more frequently than men, some authors claiming that they develop it in a ratio of two to one in the opposite sex. Pregnancy often seems to favor its development. *Heredity* is regarded as an etiological factor, but this claim is probably overdrawn, though we do observe it as a family tendency. *Congenital cases* are occasionally observed, but it is more commonly found in later life and rarely before puberty.

Pathology.—There are several varieties of simple goitre, but as they assume different pathological features, they are described in accordance with their anatomical peculiarities. A *parenchymatous* form represents a true hyperplasia of the normal tissues. The *follicular* variety is characterized by an enlargement of the follicles of the gland, while a *fibrous* goitre is due to a marked increase in connective-tissue which to a certain extent compresses the follicles. The uneven distribution of this fibrous tissue gives rise to the irregular and nodulated appearance of the gland. In cases of long standing a decided tissue sclerosis may exist. The *cystic* variety is distinguished by a follicular distention with liquid contents. Other varieties are characterized as *struma aneurysmatica* when the blood vessels are enlarged and dilated, *struma colloides,* in which the follicles are enlarged and filled with colloid matter, *struma ossea* when there is calcareous infiltration and *struma amyloidea* when there is a wax-like degeneration. Other combinations or intermediate types may be found at times and the inflammatory or suppurative process may alter the character of any of the various forms.

Symptoms.—The enlargement of a thyroid gland is readily recognized by *inspection and palpation,* while *compression* is a symptom of its later development. The swelling may vary in size from a scarcely appreciable enlargement to a large growth which will demand either mechanical support or operative relief. The enlargement may be confined to one lobe or it may affect both lobes, and the isthmus as well. The *development* is usually gradual or it may be decidedly aggravated by profound nervous irritation or pronounced menstrual irregularities. *Deglutition* may be performed with difficulty, the tumor moves vertically during this act, and a sense of fulness is an occasional complaint. *Dyspnea* may appear when the trachea is compressed, particularly during the occurrence of a cold. *Aphonia* follows from the pressure upon the recurrent laryngeal nerves. *Pain* is not a frequent complaint as the skin is not adherent and the periosteum of the neighboring bones is not compressed. The *veins* over the goitre are distended, while *headache* and somnolence are not uncommon features in connection with a large goitre. *Cerebral symptoms,* such as tetany and convulsions, are occasional complications.

Constitutional symptoms sometimes develop when the tumor has existed for a long time. Nutrition is somewhat depreciated and the inflammatory process may give rise to further constitutional manifestations. *Hemorrhage* is not an impossibility and in rare instances cretinism may be associated with a simple goitrous development. Death has also occurred as the result of compression of the vagi.

Diagnosis and Prognosis.—The diagnosis is not a matter of difficulty, inasmuch as inspection and palpation leave no doubt. Simple and malignant *adenomata* must always be differentiated. The same is true of *cancer* and *sarcoma.* When aberrant portions of the thyroid are involved intrathoracic tumors are generally in evidence. The prognosis in cases of short duration is generally favorable so far as life is concerned and a cure may be obtained if the treatment is vigorous and constant.

Treatment.—To cure a goitre will require the use of both local measures and internal medication. *Iodin* has been the favorite local remedy and as yet no better agent has been found. At first it was painted over the enlargement and absorption was encouraged by exposing the patient to the sun for a long time. Later electrolytic absorption was obtained by the use of the galvanic current after the iodin was applied. The *hypodermatic introduction* of iodin into the cystic formation has also been a favorite method of treatment with many physicians. Twenty to thirty minims of a solution containing one part of iodin to twelve parts of alcohol may be injected twice a week, providing a new point for injection is selected each time. *Electrolysis* has been employed with varying success; some practicians speak highly of it, while the majority of clinicians regard it with suspicion. *Galvanism,* if used carefully and persistently, is a reliable aid to the cure in a great majority of cases. I am quite positive that it has served me well and, though I lay claim to no special knowledge relative to its scientific use, I employ it in nearly every case which I treat.

The employment of *internal medication* has not many ardent advocates, but in my experience it is most valuable. This, with the aid of galvanism, will, in my opinion, attain a cure in the majority of cases which are not too far advanced. The remedy of first value is *iodin;* this I use in the first decimal potency and I push it to the point of stomach endurance. The same results may possibly be obtained by the use of *spongia* or other iodin preparations like *hydriodic acid* and *iodo-nucleoid.* There are cases which respond more promptly to *iodid of potash* and this, no doubt, will be true when specific complications exist. Some have used this in the third decimal potency, but in my experience the saturated solution—five to fifteen drop doses in one-half a glass of water after meals—is best. *Calcarea iodid* is naturally thought of when the special symptoms of calcarea are present; this, together with *calcarea carb.* and *calcarea fluorica,* will be called for very frequently in young girls of the chlorotic type. *Mercurius iodid, silicea* and *sulphur* will sometimes be demanded in those subjects of strumous diathesis. *Arsenicum iodid, baryta iodid* and the *iodid of antimony* may be of service in many cases. *Phytolacca* is never to be forgotten in the fibrous enlargements, particularly if it is associated with any tonsillar trouble. This remedy should be given in tincture dosage, and it is often a valuable aid to rub a strong cerate made from the same remedy into the goitrous swelling daily. *Badiaga, bromin* and *plumbum* may be of service in a few special cases. In vascular goitres we may attain favorable results in the early stages by the use of *secale, belladonna* and *hamamelis. Thyroid preparations* have been used with questionable results, as there seems to be no physiological reason for their administration.

Surgical procedure is probably undertaken too frequently for the sake of prompt removal of the tumor, and yet in the older fibrous enlargements it is probably the only treatment.

Incisions into the isthmus, tapping of the cysts and even ligature of the thyroid arteries have been undertaken with some degree of success; partial extirpation or complete thyroidectomy are the radical surgical methods which are sometimes employed.

It is essential under all circumstances to give due consideration to diet and hygiene. It is also necessary to be assured that the water supply is not a source of the disease.

MYXEDEMA.

Myxedema is a nutritional disorder which is characterized by a mucoid deposit in the subcutaneous and submucous tissue, a peculiar mental dulness and a subnormal temperature. These conditions are, without doubt, due to atrophy and the impaired function of the thyroid gland. The disease is studied under three distinct groups: true myxedema, myxedema associated with congenital or sporadic cretinism, and operative myxedema or cachexia strumipriva.

Etiology.—The majority of cases are found in girls. Heredity has been presumed to be a predisposing influence, but the real agency which leads to the atrophic change and the characteristic manifestations of the disease is not yet thoroughly understood. Neurotic conditions are probable causes, or at least they favor its development. Pregnancy is a possible factor of importance and some cases have occurred as a sequence of exophthalmic goitre.

Pathology.—It is safe to say that all forms of the disease are associated with thyroid atrophy or at least a perversion in the function of this gland. The true nature of the disease is not perfectly understood. Charcot gave it the name of *cachexia pachydermique* and believed it had a trophoneurotic origin. Therapeutic experience has demonstrated the claim that it is probably due to the loss of an essential secretion which the normal thyroid possesses. Enlargement of the hypophysis cerebri has been observed in some cases, while Horsley has conclusively demonstrated that the convolutions of the brain in these subjects are not well defined and the blood vessels are very small. This theory relative to brain perversions must be given unusual consideration, inasmuch as the cardinal symptoms of myxedema are occasionally found when the thyroid function is apparently unimpaired.

Symptoms—True Myxedema.—This will be seen most frequently in women beyond the age of thirty, though it may appear at an earlier age, and in rare instances men are afflicted. *Heredity* may be a factor, as it may attack several members of a family. *Poor food,* frequent pregnancies, prolonged hemorrhages, injuries and mental shock are liable to lead to it. The thyroid gland is invariably atrophic or perverted in its function. The *typical suggestion* of the disease is observed in the facial appearance. This is uniformly swollen, giving the "full-moon" likeness, while the skin is dry and inelastic. The normal facial expression is obliterated and the features are coarse and immobile; the nose is broad, the mouth large and folds of myxedematous tissue are seen about the neck. The patient seems to be staring constantly into space, while the physiognomy as a whole is suggestive of imbecility. *Tissue increase* is evident as the subcutaneous infiltration develops and the general bulk of the body increases; true edema, however, is not a characteristic unless it is associated with albuminuria or a cardiac complication. *Tumefaction of the skin,* however, is apparent in the face and other parts, while the mucous membranes also show the signs of infiltration. The hair falls out, the teeth may become loosened, the tongue, lips and nose are thickened and the voice assumes a peculiar tone. The *hands* are "spade-like," the feet lose their shape, while the gait is slow and deliberate.

The *nervous symptoms* are decidedly characteristic. The mind is feeble, deliberate in action and the memory is exceedingly deficient. The patient may sit for hours without giving apparent attention to his surroundings or indulging in conversation. Occasionally this hebetude may alternate with mental excitability, in which the patient is liable to be irritable or extremely violent. As the disease progresses delusions and hallucinations may develop and gradually these symptoms may terminate in dementia or melancholia. *Occipital headache* is a common complaint and occasionally cranial nerve involvement will be recognized by special sense perversions; atrophy of the optic nerve is a rare complication. A *subnormal temperature* may be observed in this disease, and the patient suffers an aggravation of symptoms during the cold weather. *Hemorrhages* may take place from the nose, gums and sometimes from the bowels. *Albumin and sugar* occasionally appear in the urine and ascites is rarely noticed.

MYXEDEMA ASSOCIATED WITH CONGENITAL OR ACQUIRED CRETINISM.

The symptoms under this head will be typical of myxedema in childhood. In other words, the true expression of the disease refers to an *arrested development* which pertains to the mind and body alike. The true picture of a cretin is that of a dwarf. The *congenital form* is characterized by the absence of a thyroid gland, the thick neck, short arms and legs and the prominent abdomen. All of these patients have large heads and faces, thick lips, together with thick and protruding tongues. All the bones are short and broad, while the epiphyses are swollen but not ossified. In some respects the cretin resembles a rickety child. The subcutaneous tissues show the signs of myxedematous infiltration; the growth of the child is retarded and the mentality is that of idiocy. Speech is imperfect and unintelligible, the power of locomotion is rarely attained, anemia is often very profound and early death from intercurrent disease may take place. Fortunately true congenital cretinism is a rare disease.

Acquired cretinism may start so near the time of birth that it is hard to differentiate between this and the congenital form; usually it is recognized between the second and fifth years. The physical development is arrested to the extent that the child does not assume the proportions commensurate with his advancing years. In addition to this we shall observe the prominent symptoms which characterize true myxedema.

Endemic cretinism has been noted on the continent of Europe in localities where goitre is also endemic; it is most common in the mountainous districts and it may be associated with goitrous enlargements, though a fair proportion of endemic cretins are without goitre. When goitre does exist in these cases we may ascribe it to an overgrowth of the fibrous tissue at the expense of the glandular formation. *Sporadic cretinism* is found in England and America and these cases are usually characterized by an atrophic thyroid. In both the sporadic and endemic cases the normal function of the gland is destroyed. The arrested development starts in conjunction with the thyroid inability and, though the cretin may live to the age of thirty or forty, the physical development generally ceases before the age of twenty. The typical symptoms of myxedema prevail in both the congenital and acquired forms of cretinism.

Operative Myxedema.—This is a rare form of myxedema which appears as the result of complete surgical removal of the thyroid gland. It rarely follows a partial removal of the gland, though this is possible when the function is destroyed thereby.

Diagnosis.—This is made with no difficulty, inasmuch as the symptoms are so clearly indicative of the disease. The edema of heart disease or nephritis will be easily differentiated unless there are complicating diseases. The sluggish mental action, combined with the characteristic objective symptoms, will be clearly recognized by a careful observer.

Prognosis.—Until recently the disease was regarded as incurable. The introduction of the thyroid extract has fortunately removed that prognosis. If a typical case is taken under treatment in time a cure may be obtained in the average case. This is to a certain extent true of cretinism, though the congenital cases are necessarily considered as incurable. Death may follow from some other cause.

Treatment.—*Hygienic detail* is an early essential of the treatment. A warm climate is always conducive to the favorable action of remedies. The body warmth must be maintained by sufficient clothing, massage and warm baths. In like manner it will be necessary to institute a judicious but nourishing diet. *Thyroid feeding* has placed the disease within the control of the physician. The sheep's thyroid is the preferable preparation, and it is used either in a glycerin extract or in tablet form. This must be administered with great care and the minimum dose should at first be employed, as gastric irritation and cardiac depression are dangerous possibilities when the remedy is crowded too fast. To further guard against cardiac exhaustion, it will be best in the early treatment either to place the patient in bed or at least curtail the physical exertion. The cumulative action of thyroid extract may be noticed in the treatment of some patients; this will be determined by the appearance of vomiting, renal pain, tachycardia, cyanosis, vertigo or depressing headaches. When any of these symptoms persist the remedy should be discontinued for awhile. When toxic action arises from the use of thyroid preparations it is usually manifested in the early stages of treatment. The *dosage* of the glycerin extract should be ten minims daily at the start and gradually increased to effect and then reduced until the patient finally takes one dose per week. The powdered gland or tablets may be used in like manner, starting with three to five grains daily. Caution should be observed lest an impure product be employed. The *homeopathic practician* has been taught the efficacy of a smaller dosage, and with this theory I heartily concur. The first, second and possibly the third decimal potency may be administered, with increased frequency, and invariably a better result is obtained. This opinion I have confirmed in the cases which I have been privileged to treat.

Other remedies are also to be considered from their symptomatic standpoint. *Arsenic* should receive first attention, and I have in my records a typical case which responded to the persistent use of this remedy and he is now a well man. In this case the hebetude was so pronounced that the patient took to his bed for over a year, but is now—six years later—attending to his business without any evidence of his previous affliction. Some regard the conjoint action of arsenic and thyroidin as unusually favorable. *Strychnia* has been employed with some degree of success, while *pilocarpin, atropin, amyl nitrite, physostigma* and *lycopus* may be considered for their symptomatic peculiarities.

NEOPLASM OF THE THYROID.

The thyroid gland may suffer also from a variety of morbid growths. *Adenoma* may exist in the form of encapsulated nodules in one or both lobes, or it may assume the dimensions of a large sized growth, supplanting to a considerable extent the normal thyroid structure. *Primary medullary cancer* may develop from the epithelial cells of the thyroid follicles and *secondary cancer* is occasionally observed. *Tuberculous goitre* is a rare finding, while neoplasms may sometimes develop in the thyroid as the result of *syphilis, hydatid disease* and *actinomycosis.* The treatment of these belongs chiefly to the surgeon.

DISEASES OF THE RESPIRATORY SYSTEM.

DISEASES OF THE NOSE.

ACUTE RHINITIS.

This disease is also referred to as acute nasal catarrh or acute coryza. It is characterized by a catarrhal inflammation of the nasal mucous membrane extending also to the adjacent sinuses and passages.

Etiology.—The disease is usually attributed to sudden changes in the weather and exposure to draughts, and it may also result from the inhalation of irritants. Since it may occur in the epidemic form and so often spreads to other persons of a family, it is therefore probable that the cause is a microorganism. There is no doubt in my mind that autointoxication is a large predisposing factor.

Symptoms.—A *sense of chilliness,* slight temperature (100°-101°F.), headache attended by pain in the back and limbs, and sneezing usually initiate the attack. The *pulse* is generally quickened, the surfaces are at first dry, thirst may be excessive, while anorexia and constipation are usually present. The *mucous membrane* of the nose is decidedly swollen and hyperemic, causing the sensation of being "stuffed up" and obstructing respiration. There is usually impairment of taste and smell. With this there may be soreness of the throat, stiffness of the neck, difficult deglutition and hoarseness. Hearing is also usually impaired as the result of inflammatory extension into the Eustachian tubes. There may be a coexisting bronchitis, which gives rise to a slight cough, and the infection may be evidenced by the appearance of labial or nasal herpes. The thin and irritating *secretion,* which soon becomes muco-purulent in character, is the most characteristic feature of the disease. The *duration* of the disease is usually limited to four or five days, the nasal discharge and other symptoms gradually disappearing.

Diagnosis and Prognosis.—The *diagnosis* of this disease is not usually difficult, though care must be exercised to avoid confusion with measles, influenza and other diseases in which coryza is but a symptom. The *prognosis* is always favorable so far as recovery is concerned, but complicating sinus disease or bronchitis may add to its duration.

Treatment.—The majority of these cases will require very little treatment other than keeping the patient in bed for a few days. Elimination must be encouraged by augmenting bowel, kidney and skin secretions. The hot bath, a limited liquid diet and plenty of pure drinking water are essential features of treatment. *Aconite* frequently is the only remedy required, and is indicated by the chilliness, fever, sneezing and dryness of nose and also of the skin. Closely following aconite we may find that *belladonna* is required or when the initial fever is slight and the cerebral congestion is marked it may be the first indicated remedy. I usually use the first or second decimal potency and I give the medicine in the early stages as often as every half hour until there are signs of improvement. *Allium cepa* is indicated in the initial stage when there is a profuse and thin discharge from the nose and an attending profuse lachrymation. The use of raw onions as a diet in the early stage may assist the remedy materially. *Camphor* in the lower potencies is often successfully used to abort the disease in its early stages. *Sambucus nigra, gelsemium, ambrosia, pulsatilla* and *natrum mur.,* may be indicated in place of these. As the discharge becomes muco-purulent we shall need to employ such remedies as *arsenicum iodid, kali bichromicum, dulcamara, hepar sulphur, sulphur, sanguinaria, mercurius corrosivus* and *hydrastis.*

The *local treatment* of this disease is a popular one and gives much relief. It consists in the frequent use of sprays containing menthol, pine needle oil, eucalyptus, sodium sulphocarbolate and other cleansing and soothing agents. A few crystals of menthol dropped into boiling water with inhalation of the steam is of temporary benefit, and when headache, due to closure of the sinuses is predominant, hot compresses over the nose and eyes is acceptable to the patient. The inhalation of steam from a pint of water containing one dram of the compound tincture of benzoin is of value.

CHRONIC RHINITIS.

Chronic nasal catarrh is usually recognized in two forms, the atrophic and hypertrophic, though the pathologic features of each may be recognized in the same case. It is commonly a sequela of the recurrent acute form, though many diseases of an infectious character may have a relation to it, if they are not the real causative agents. Some have regarded the atrophic disease as purely of infectious character, the bacillus mucosis ozenae of Abel being ascribed as the bacteriological cause.

In *hypertrophic rhinitis* the pathologic change is characterized by redness and swelling of the nasal mucosa which covers the turbinal bones, and an increase in the tissues. As the membrane thickens there is a resultant obstruction of nasal respiration, together with increased secretion from the mucous membrane. This is thicker than normal, due to inactivity of the mucous glands, hence less evaporation by normal inhalation and therefore a retained secretion. In *atrophic rhinitis* the nasal cavities are enlarged and the mucous membrane is covered with a thick, yellowish-green exudate which, after decomposing, is seen in the form of a crust. A horrible odor is emitted from the nose, which makes the disease decidedly loathsome, though the patient is usually ignorant of that fact, because the olfactory nerve endings are destroyed. The atrophic form of this disease is frequently a sequence of the hypertrophic form and the pathologic change may reach the stage of ulcerative degeneration.

Symptoms.—In the *hypertrophic form* of rhinitis the most characteristic symptom is impairment of nasal respiration on account of the hypertrophy of the mucosa covering the turbinated bones. There is a posterior nasal secretion which may induce a constant cough with an attempt to clear the throat. The sense of smell is lost and other disturbances, which are due to the nasal obstruction and pressure, are present. In the *atrophic form* the only obstruction to the nares is due to the presence of the nasal crust. The passages usually show an increase in the size of the chambers as the result of the atrophy. Because of this atrophy, which involves the mucous glands, there is marked dryness due to absence of their secretion.

Treatment.—In hypertrophic rhinitis a cleansing solution of soda bicarbonate and common salt in equal parts, or a saturate solution of sulphocarbolate of soda is usually required. Other slightly antiseptic and alkaline solutions may be employed by means of a spray. Powders have been discarded and the nasal douche should never be used in this form of rhinitis. The indiscriminate permission which is often given to patients to treat themselves in this manner is very dangerous, owing to the frequent carrying of infection to the middle ear and to the mastoid. Nasal douches are only applicable in the atrophic form of rhinitis and then only with the utmost regard for the patency and location of the eustachian orifice. Patients should be cautioned not to blow the nose after using either sprays or douches and not to go out of doors for at least half an hour. The removal of hypertrophied tissue is of greatest importance. This may be done by the local application of chromic or trichloracetic acid or by means of the galvano or thermocautery, but better still are the cleaner and more exact cutting operations to correct deflected septa, and remove pronounced enlargements of the turbinated bones by those who have had experience in this work. The object in the treatment of this form of catarrh is to give a free and healthy passage for inhaled air and to provide drainage for the secretions.

In the *atrophic form* not so much is expected from local treatment except by the continued use of cleansing sprays to overcome the catarrhal discharge and to correct the foul odor. Dobell's and Seiler's solutions, preparations of carbolic acid and peroxid of hydrogen, are serviceable in this respect. These watery sprays should be followed by oily solutions such as menthol in albolene (grs. 10-20 to the ounce), both to protect the membranes and to overcome the foul breath. After the removal of crusts stimulation by solutions of nitrate of silver (grs. 20-40 to the ounce) may help to promote healthy granulation.

Internal remedies are of service so far as they pertain to the pathologic cause and the inflammatory condition which produces the nasal discharge. The diathesis is a matter for consideration in the selection of a remedy. For the fetid catarrh we have several valuable homeopathic remedies. *Hydrastis, iodid of arsenic, kali bichromicum, pulsatilla, argentum nitricum, calcarea iodid, aurum chlorid, mercurius, nitric acid, graphites, hepar sulphur* and various other remedies may be mentioned and should be studied.

HAY FEVER.

Hay fever, vasomotor rhinitis or autumnal catarrh, is a form of coryza produced by the intense swelling of the nasal mucous membrane during certain periods of the year.

Etiology.—Many causes have been given for the existence of this recurrent form of nasal catarrh, but as yet no micro-organism has been discovered. The pollen of the ragweed, golden rod and other flowering plants have been mentioned as the irritating factors. When it occurs in the spring of the year it has been attributed to the pollen of roses and hence the term "rose cold." Sudden changes of temperature, nervous causes, heredity and certain climatic influences have also been claimed as etiological factors. Lithemic subjects are particularly prone to attacks. Anatomical changes in the nasal passages, such as mucous membrane hypertrophy, deflection of the septum, nasal spurs, diseased turbinates and polypoid growths are often causative factors. Reflex irritations and neurotic constitutional conditions have been regarded as predisposing influences.

Symptoms.—The *local symptoms* are characterized by an abrupt onset at a certain time of the year. Coryza attended by sneezing, nasal obstruction and a profuse watery discharge typifies the early symptomatology. In addition to these symptoms there is usually conjunctival inflammation, lachrymation and marked itching of the naso-pharynx, while the sense of smell and taste are usually impaired. Exacerbation of the catarrhal features generally occurs during sudden changes in the weather or after exposure to damp air. Bronchitis may be an associate complication whenever the bronchial mucous membrane is invaded by inflammatory extension. *Constitutional disturbances* are frequently present. The most marked of these are the tendency to chilliness and fever, anorexia, insomnia, aprosexia and general physical exhaustion. The disease generally lasts two to six weeks, a cure often being affected only by a change of climate or the appearance of frost.

Diagnosis and Prognosis.—The *diagnosis* is made by the suddenness of the onset and the catarrhal symptoms just described. The fact that it occurs at a definite time each year is a guiding clinical symptom. The *prognosis* is favorable so far as life is concerned, but a cure by medical means is not easily accomplished. The disease frequently is associated with asthmatic attacks.

Treatment.—*Local treatment* has been assiduously employed with the hope of relieving this trouble. So far no specific medication has been discovered which has accomplished much in this respect. Cocaine (1 to 4 per cent) has been frequently resorted to for the relief of the catarrhal irritation, but this is an agent which has fortunately been condemned of late. Boric acid (10 grs. to 1 ounce) has been employed for the cleansing of the parts and for the conjunctival involvement. Irrigation of the nasal passages, with normal salt solution, or a weak solution of quinin, has been recommended. Daily cleansing of the nasal chambers with Dobell's solution has been favored by some, while the oily preparations, administered by an inhaler or by direct application, are also popular agents. Subnitrate of bismuth and boric acid (1/2 dram) to the ounce of vaselin, has been suggested as an application to relieve the intense itching. Adrenalin chlorid gives immediate relief in many severe cases, but it is questionable how permanent such results may be, and it is possible that unfavorable reactions may recur from its persistent use. Pollantin, an antitoxin procured from the serum extracted from a previously prepared horse, is being used successfully. The medicated powder is snuffed up the nose and dusted into the eyes and relief follows in a short time and a cure in many cases.

Internal medication has given very little satisfaction so far as our present knowledge is concerned. *Atropin* has been employed (grs. 1/300-1/200), both internally and hypodermatically, with the object of inducing dryness of the nasal mucous membrane. *Iodid of potash, belladonna, morphin* and *chloral hydrate* in gradually increasing doses have been suggested, but these alike are dangerous and do not generally accomplish the object desired. The *iodid of zinc,* the *arsenite of potash* and the *arsenate of quinin* have been recommended by Goodno.

Among the homeopathic preparations we should first mention *arsenic,* which has a great influence upon the mucous membrane of the nares and conforms closely to the prominent symptoms of the disease. The *arsenite of copper, arsenicum iodid* and the *arsenate of strychnia* should not be overlooked. *Arundo mauritianica,* a new remedy within three years, has been frequently of benefit. For the asthmatic phase of the disease *grindelia robusta, ipecac, terebinth, sanguinaria, euphorbium, pulsatilla* and *lobelia* may be remembered. In severe cases atropin (grs. 1/300-1/200) may be given hypodermatically two or three times daily. Sometimes *strychnia nitrate* (grs. 1/60-1/30) hypodermatically administered, gives relief for some time. *Sabadilla* often helps the coryza with attendant conjunctival disturbance and *ambrosia* is a remedy which has the exact symptomatology of this disease. *Sticta* is spoken of with favor when there is an associate cough the result of laryngo-tracheal irritation. *Allium cepa, gelsemium, euphrasia* and other remedies have received attention in the treatment of this disease, though as yet we are obliged to state that no remedy has given entirely satisfactory results.

EPISTAXIS.

Nosebleed refers to a hemorrhage occurring within the nasal cavities. It may be due to intense hyperemia or ulceration of the nasal mucosa, or it may arise from the presence of polypi or other foreign growths. Injury may be the cause or a constant picking of the nose may induce it. It is also a feature of certain diseases such as typhoid, influenza, pernicious anemia, leukemia and hemophilia. Many are predisposed to it by a natural plethora, it may appear from overexertion when arteriosclerosis is present and it is likewise a symptom of vicarious menstruation.

Symptoms.—The flow of blood from the nose usually occurs by constant dropping, though in some conditions, like hemophilia, it is liable to gush for a time before any relief is obtained. The blood may flow into the pharynx and be expectorated or it may be swallowed and give rise to the vomiting of blood. In either case fears are liable to be aroused relative to the possibility of tuberculosis or gastric ulcer. When the blood has its exit from the anterior nares this doubt in a diagnosis will not exist. In all cases a rhinoscopic examination is the only means by which the location of the bleeding point can be determined. No secondary symptoms usually follow as the result of nosebleed, as in most cases it ceases because of coagulation. In prolonged hemorrhage exhaustion is liable to be experienced. When it is a sign of fracture of the skull at the base a fatal result may be expected.

Treatment.—When the epistaxis is due to a local cause, treatment should be applied to arrest this, though in the majority of cases, this will be done spontaneously. By means of proper illumination and a nasal speculum it is usually possible to find the bleeding point and the hemorrhage may then be stopped by slight pressure with cotton pledgets soaked in hydrogen peroxid or adrenalin or cauterization with silver nitrate or chromic acid. The injection of cold or very hot water into the nares or such astringents as tannic acid, alum, lead and zinc also will usually suffice. Perchlorid of iron, diluted with cold water, is an old remedy. At present we have a most serviceable local agent in adrenalin chlorid. Often the application of ice, externally or at the back of neck, will aid in stopping hemorrhage, but in some cases it may be necessary to plug the posterior nares with gauze tampons drawn forward from the nasopharynx by a catheter; or pack with narrow strips of lint impregnated with bismuth subnitrate through the anterior nares, each strip being pressed down firmly and smoothly, building from the floor upward.

Internal remedies are not always needed, but we have many which certainly help to correct the hemorrhage and in many instances they will suffice without the aid of local measures. *Aconite, ipecac, hamamelis, arnica* and *secale,* in the third and lower potencies, are valuable agents in the early stages of epistaxis. *Phosphorus, china, nitric acid, crocus sativa* and *crotalus horridus* are useful in the recurrent hemorrhage and when there is some underlying constitutional predisposition. When uterine conditions are factors in epistaxis *erigeron, trillium, sabina* and *ustilago* may be indicated. In young children we may obtain good results with *ferrum phos., calcarea carb.,* and *veratrum viride.* Goodno calls special attention to *ferrum muriaticum* in the first decimal dilution or in drop doses of the tincture. He has also recommended *hydrastin hydrochlorate,* in the second or third decimal potencies, for frequent and serious epistaxis.

DISEASES OF THE LARYNX.

ACUTE LARYNGITIS.

This is an acute catarrhal inflammation of the laryngeal mucous membrane. It is either an independent affection or it may be due to a general catarrhal inflammation of the upper respiratory passages. It is frequently contracted by exposure to cold or getting the feet wet, and in many instances it is a family predisposition. The general physical condition is a factor of importance, for those suffering with a natural debility are predisposed. Those who are inclined to take too much care of themselves and do not develop a healthy physical resistance easily contract the disease. The habit of wearing too heavy woolen underclothing and excessive outer wraps often favors it, as does a lack of body cleanliness and proper elimination. The inhalation of irritating vapors or gases or the local effect of corrosive poisons or in fact any agent which induces hyperemia of the laryngeal mucosa may develop the disease.

Symptoms.—One of the first symptoms which suggests the disease is a marked *hoarseness* which soon develops into complete aphonia. The larynx is irritated by the inhalation of cold air or even by the normal respiratory act. A *dry and irritating cough* is also an early symptom and if there is much edema of the laryngeal mucosa, this may be attended by dyspnea. The patient often complains of the presence of a foreign body in the region of the larynx. When the secretions are freely eliminated the cough is greatly relieved.

A *laryngeal examination* reveals a swollen, tumefied and reddened mucosa, the vocal chords and the aryteno-epiglottidean folds partaking of the inflammatory change. As a result of this inflammation the vocal cords lose their mobility, thus causing aphonia. The epiglottis and the trachea may partake of the general inflammatory extension.

Diagnosis.—A laryngoscopic examination will explain the symptomatology though the loss of voice may simulate a condition of nervous aphonia. It is often difficult to use the laryngoscope in children and thus it is essential to perfectly interpret the symptoms. When a proper examination can not be made it may possibly be confused with edema of the glottis. *Laryngismus stridulus* is differentiated by the absence of fever and coryza and by the suddenness of the onset. *Membranous laryngitis* will be distinguished by the more pronounced constitutional depression and the enlargement of the cervical lymphatics.

Prognosis.—The prognosis is usually favorable if the patient is given proper care and responds readily to the use of indicated remedies. When the disease is associated with serious complications or when marked edema occurs, a more unfavorable prognosis should be given. Neglected cases are liable to terminate in chronic laryngitis. Cases of laryngitis lasting more than two weeks are usually infective.

Treatment.—*Rest* of the inflamed larynx is quite essential and for this purpose the patient should be placed in bed, no company being allowed in the room, except the regular attendant. Talking should be prohibited and whispering allowed only when necessary. Smoking is not allowed. The *room temperature* should be maintained at an even degree and inhalations of moist air are often favorable to the relief of laryngeal irritation. This may be attained in many ways, a simple measure being to fill a cup or pitcher with boiling water, into which one or two drams of compound tincture of benzoin is placed. This may be placed near the bed or the patient may inhale the steam directly by means of an inverted funnel. *Steam atomizers* are convenient for this purpose, into which eucalyptus, pine needle oil and similar oil preparations may be placed. An *icebag* or the early use of external cold packs about the throat are often sufficient to suppress the inflammatory invasion in the first stage. *Hot applications* are also advised by many practicians of experience. External application of such cerates as phospho-tar ointment, Trask's ointment and even "goose oil" may afford some palliation. Sipping of hot milk aids in the expectoration of mucus from the larynx.

Homeopathic remedies administered according to their specific indications furnish our most reliable means of treatment. *Aconite* is invariably indicated in the first stage and this is often sufficient to accomplish the cure in many cases. *Iodin, bromin, spongia* and *benzoin* are our best remedies next to aconite and these are usually employed in the third decimal potencies. In case of prolonged inflammation or extreme exudation, *calcarea iodid* or *iodid of arsenic,* in the third decimal potency, may be required for several days. In line with these remedies we may compare *kali bichromicum, hepar sulphur* or *kali iodid.* In case of persistent aphonia we shall have use for *phosphorus, causticum* or *ammonium mur.* When gastric disturbances are associated, we may have reason to prescribe *ipecac, ignatia, pulsatilla* or *lobelia.* When the catarrhal symptoms are associated with coryza, *ambrosia, sam-*

bucus, eucalyptus and *sanguinaria* may be indicated. In attacks of acute laryngitis the nervous phase of the disease is often recognized. In such instances *belladonna, hyoscyamus, stramonium, gelsemium, agaricin, camphor monobromated, asafetida* and *moschus* may give relief.

CHRONIC LARYNGITIS.

This disease usually follows repeated attacks of acute laryngitis. Overuse of the voice may induce it and it is, therefore, common among public speakers. Other causative influences may be ascribed to the constant use of tobacco, immoderate indulgence in alcoholic stimulants or the inhalation of dust-ladened air. The laryngeal mucosa is reddened and thickened, with occasional erosions which may terminate in ulceration. The mucous glands are prominent; villi may project in the form of nodular swellings and, in rare instances, there are marked changes in the vocal cords. Vesicles are rarely observed upon the laryngeal surface, giving rise to a form of herpetic laryngitis.

Symptoms.—The early signs of chronic catarrhal laryngitis are characterized by *repeated laryngeal irritation* and attacks of hoarseness. These symptoms are generally enhanced by any attempt to use the voice beyond its natural limitation or by the prevalence of damp weather. The patient constantly tries to clear the voice by "hemming and hawking" only to increase the hoarseness. A sense of throat irritation is always present and the voice can not be used in the open air without inciting the disturbance. The *cough* shows much variation in degree, varying from the short and irritating "hack" with laryngeal tickling, to the more extreme paroxysms. It is usually dry, but a mucus or catarrhal expectoration occasionally results from the irritation and low grade inflammation. *Pain* is not usually a factor, though this may be incited by additional colds or the inexcusable efforts to use the voice when the hoarseness exists. A laryngeal examination may reveal the reddened mucous membrane with the destruction of the mucous glands.

The *diagnosis* is not obscure when the laryngeal examination reveals the pathologic condition which incites the symptoms. The *prognosis* is favorable as the majority of cases are amenable to persistent and systematic treatment. The tendency of the disease is to pursue a chronic course and the persistency of the chronic inflammation may predispose to tuberculous development or malignant changes.

Treatment.—The first requirement in treatment is to insist upon the patient giving the voice a *rest*. This will often allay the chronic irritation, but a climatic change is frequently necessary to perfect a cure. The use of the atomizer with medications for the *local cleansing* and palliation of the irritated parts, is a necessary feature of the treatment. For this purpose it is well to mention the value of the sodium salts, the biborate, the bicarbonate, the salicylate, etc. The care of the *general health* is of the greatest importance and the hygiene must be thoroughly looked into. Such patients often need a tonic as well as a sea voyage. This may be obtained by means of the usual tonics such as iron, quinin, chinoidin, arsenic, strychnia, or preferably by the indicated homeopathic remedy. Often the elimination is entirely at fault and if a thorough salicylate treatment is carried out the throat trouble may quickly respond to the indicated remedy. While I do not favor the too frequent employment of cathartics, particularly calomel, I am obliged to admit that there are cases which are in need of just such agents. No remedy will work as we desire if the elimination is deficient. This is particularly true of chronic throat diseases and we should encourage elimination by the use of an abundance of salines and occasionally by a brisk cathartic.

The *local treatment* will include many astringent solutions which are to be used by means of a spray. Alum (grs. 15 to the ounce of water), tannin (grs. 5 to the ounce), zinc sulphate or chlorate of potash (grs. 15 to the ounce), and sulphate of iron and ammonia (1/2-1 dram to the ounce) are frequently employed. Before these solutions are used the mucous membrane should be cleansed by an alkaline spray. Steam inhalation may be required the same as in the acute form, a teaspoonful of compound tincture of benzoin being added to a pint of boiling water. Lozenges of benzoic acid (in each gr. 1/2) or cubebs (gr. 1/2), ammonium chlorid (grs. 3-5) are both serviceable and convenient for temporary relief. Many of the preparations put out by our reliable pharmacies may be of service.

The *medicinal treatment* has heretofore included such remedial agents as codliver oil, creosote and expectorants, none of which is of much value in this disease. The homeopathic remedies will meet the pathologic conditions with better success than any other measures. Among those most frequently indicated we shall find many already enumerated in the treatment of the acute form of this disease. The most prominent of these are *phosphorus, causticum, hepar sulphur, hydrastis, esculus, collinsonia, kali bichromicum, argentum metallicum, argentum nitricum, arsenicum iodid* and *nitric acid. Manganum* has been recommended in chronic laryngitis occurring in connection with pneumonia or tuberculosis.

SPASMODIC LARYNGITIS.

Spasm of the glottis, or false croup, is generally known as laryngismus stridulus and usually occurs in children under the age of five. It is probably of neurotic origin, though it may be associated with an acute catarrhal condition. It is commonly observed in rachitic children and in those with adenoids and enlarged tonsils. Occasionally an elongated uvula will cause attacks very similar to this disease. Exciting causes predispose and among them we may mention the sudden attacks of acute indigestion, cardiac diseases, nasopharyngeal diseases, bronchial or tracheal inflammations and gastrointestinal derangements. The attack is caused by a spasmodic contraction of the adductors of the vocal cords temporarily closing the glottis and preventing inspiration. Syphilis is believed by many to be an underlying cause.

Symptoms.—The *nervous type* has no relation to a catarrhal inflammation of the larynx. It is characterized by the sudden spasm which induces dyspnea and facial cyanosis which is very alarming. It usually appears without warning during the night and is manifested by the sudden arrest of respiration, upon the cessation of which there is a high-pitched "crowing inspiration." These paroxysms are occasionally repeated several times within twenty-four hours and general convulsions are sometimes present, though there is usually no fever nor other signs of laryngeal inflammation.

The spasms which are *associated with catarrhal laryngitis* have a similar but not so severe a symptomatology. In these the child usually wakes suddenly at night with a croupy cough which is attended by a slight laryngeal spasm. There is some evidence of cyanosis and dyspnea, but all of these acute symptoms soon subside. This attack soon closes and in the morning the child is apparently well, only to have a repetition of the attack on the succeeding two or three nights.

Diagnosis.—The suddenness of the attack and the short duration of the spasms and the slight fever, cough and aphonia are the diagnostic features. In membranous laryngitis the more gradual onset which has no marked intermission is a differentiating symptom. *Laryngeal diphtheria* will be recognized by the bacteriological examination and the evidence of a profound toxemia.

Prognosis.—A serious result rarely occurs in spasmodic laryngitis if prompt attention is given to the patient. The condition of the nervous system and the general physical state will demand systematic treatment. Any attendant catarrhal inflammation of the larynx will also require careful attention.

Treatment.—The best treatment of the emergency is the *hot bath* (temp. 98°-110° F.) to which may be added a handful of mustard. Hot water may be constantly added until perfect relaxation takes place. The epiglottis can be drawn forward by traction on the tongue. The use of an *emetic* may be of value, the syrup of ipecac (drams 1/2-1) repeated every ten or fifteen minutes, until free vomiting takes place, will often cut short the attack. Sometimes the simple irritation of the fauces with the finger will induce this. Powdered alum, given with molasses or honey, will also be serviceable. *Free admission of air* by the wide open window and the dashing of cold water into the child's face while in the semi-recumbent position may overcome the spasm. *Hot moist applications* applied externally to the larynx are also successful in mild cases. The external application of an icebag in the region of the larynx or over the epigastrium, the inhalation of chloroform or amyl nitrite, are also recommended.

During the *intervals of the paroxysms* attention should be given to the general physical condition and the nervous state. These are best attended to by the use of a well indicated remedy. Should there be any abnormal condition such as adenoids, enlarged tonsils, etc., they should be removed. *Internal remedies* are not generally prompt enough in their action for the treatment of a spasm. *Aconite,* however, will usually cut short an attack when given frequently till effect. *Cuprum metallicum, hyoscyamus, ipecac, ignatia, chlorin* and *moschus,* in the second and third decimal potencies, have been highly recommended and are no doubt of service in many cases. *Sambucus* has been highly praised by Goodno, and it certainly corresponds to the cardinal symptoms of the disease. *Belladonna, gelsemium, kali bromatum, valerian, asafetida* and *sumbul* may be suggested by the clinical picture. During the interparoxysmal state we shall find use for any remedy which covers the symptom-complex and the general history of the case. *Arsenicum iodid, calcarea carb.,* and *iodid, baryta carb.,* and *iodid* will usually meet the constitutional condition.

TUBERCULOUS LARYNGITIS.

It has been conceded of late that primary laryngeal tuberculosis is a possibility, though it is a very rare disease. Tuberculous laryngitis is usually secondary to pulmonary tuberculosis as the larynx does not furnish a very favorable soil for the development of the tubercles. As a secondary complication the infection is probably developed by the expectoration of tuberculous sputum from the lungs. It is claimed that 25 per cent of all cases that die of pulmonary tuberculosis at some time have tuberculous laryngitis.

Pathology.—Tubercles are observed in the swollen mucosa of the larynx in the neighborhood of the blood vessels. These have a tendency to fuse into small tuberculous masses which undergo the secondary changes of caseation and ulceration. The mucosa is greatly thickened, particularly about the arytenoid cartilages and the ulcers are covered with a grayish exudate. As the result of the bacillary invasion the vocal cords may suffer an erosion while a perichondritis may follow with necrosis and exfoliation of the cartilages. The disease may extend into the pharynx and esophagus while the tonsils and epiglottis are frequently involved.

Symptoms.—The *early symptoms* simulate those of catarrhal laryngitis, but a suspicion of tuberculosis should be established by their intractability. A slight *huskiness of the voice* sooner or later develops into a marked hoarseness which may become so extreme that complete aphonia results. An *ineffectual cough* is another prominent symptom and about the same time there is usually a pronounced dysphagia. This is marked when the epiglottis is implicated or the ulceration extends into the pharynx. The laryngoscope will reveal the thickened and infiltrated mucous membrane, particularly over the arytenoids. The *tuberculous ulcers* "are broad and shallow with gray bases and ill defined outlines." A similar thickening with ulceration may be observed on the vocal cords. *Inanition* and *emaciation* finally result, due to the inability to take sufficient nourishment. Besides the local symptoms fever, night-sweats and cough may be observed.

Diagnosis.—Frequent attacks of laryngitis without obvious cause, or attacks lasting more than two weeks, should suggest some form of infection, and usually laryngeal tuberculosis. When any or all of the foregoing symptoms are present the patient should be given a careful examination of the chest and larynx. This symptom-complex can be due to nothing else than tuberculosis. *Syphilis* will not present the evidence of pulmonary involvement, while the tuberculin test and the iodid test will establish the differentiation. Both syphilis and tuberculosis may affect the larynx at the same time. Malignant growths are to be differentiated by their appearance, usually after the forty-fifth year, by the darker color of the tissues involved, which contrasts vividly with the paleness of the tuberculous lesion, by the severe pain which precedes ulceration, and by the enlargement of the lymphatics which is not so pronounced nor so constant as in tuberculosis.

Prognosis.—Under most circumstances the prognosis is grave. Primary tuberculosis of the larynx may be more hopeful, but the disease is at best a serious one. Death occurs in the majority of cases in from three to twelve months. When the disease is recognized early and treated vigorously, hope may be entertained.

Treatment.—The measures suggested for local treatment in catarrhal laryngitis may be employed at least for temporary relief, and the same is true of internal remedies. Solutions of tannic acid, silver nitrate and cocain have been advised for the relief of pain. The technique of treatment is, however, so difficult that only a specialist is able to do this work with success. Morphin internally and in the form of an insufflation with starch or even as a spray has been recommended, but this, too, is only temporizing. *Iodoform,* applied in the same way, is probably more efficacious. The general treatment for tuberculosis must also be relied upon. *Iodoform, iodin, phosphorus, bromin, arsenicum,* the *iodids of arsenic, calcarea, stannum, argentum nit., drosera* and *causticum* are worthy of a trial according to their indications. Cocainization of the larynx followed by the skilful application of lactic acid, 25 per cent increased to 70 per cent, has cured some cases. The *diet* should be as nutritious as possible, while life in the open air is of great value.

SYPHILITIC LARYNGITIS.

Syphilis frequently invades the larynx and may be a manifestation of the acquired or congenital form. The acquired may occur as primary, secondary or tertiary lesions. The congenital usually appears within the first eighteen months of life. The pathology consists of erythematous patches, mucous patches, gummata and ulcers located most frequently on the epiglottis, vocal cords, epiglottic folds, ventricular bands and on the posterior wall of the larynx.

Symptoms.—In the *secondary form* the symptoms are defined by an erythema and catarrhal condition, associated with laryngeal irritation and hoarseness or slight aphonia. Superficial white ulcers are symmetrically located on the cords or ventricular bands, while mucous patches occasionally occur and condylomata are rarely seen. In the *tertiary form* the gummata and deep ulceration may lead to serious stenosis or, by the process of necrosis and exfoliation, destroy portions of the cartilage or create hemorrhage by arterial erosion. Painful deglutition, dysphagia and a sensation of a lump in the throat are prominent symptoms. Hoarseness and aphonia are present in varying degree. In *inherited syphilis* the symptoms appear during the first year of the child's life or rarely about puberty. Gummatous infiltration with secondary ulceration will be noticed most commonly upon the epiglottis or in the ventricles, giving rise to cicatricial contraction or invasion of the cartilage. Accompanying the local lesion of laryngeal syphilis it is usually possible to find other signs of the specific infection or inheritance. Among the more prominent symptoms of this form is the peculiar characteristic "bleating, almost voiceless, cry of the child." Respiration is obstructed, edema of the larynx may occur, necessitating tracheotomy, and laryngismus stridulus is of frequent occurrence. The ulceration may cause dysphagia, interfering with the nourishment of the child.

Diagnosis and Prognosis.—The diagnosis will depend not alone upon the symptoms just enumerated, but will depend also upon the history and other signs of the specific invasion. *Tuberculous laryngitis* is usually associated with the signs of pulmonary tuberculosis and it is possible to discover the bacilli in removed portions of the diseased tissue. *Carcinomatous laryngitis* presents a nodular proliferation as compared to the destructive process of the syphilitic lesion and the ulceration of the former is single. The *prognosis* is fairly favorable if antisyphilitic treatment is undertaken early. Stenosis is the most unfavorable result. In children under two years of age the prognosis is bad, few of them surviving.

Treatment.—The internal use of iodid of potassium and mercurial inunctions are always demanded. Other internal medication should be given according to symptomatic suggestions. Topical treatment is beneficial the same as in catarrhal laryngitis and strong solutions of nitric acid or silver nitrate are often required to cure the ulceration. Such remedies as *nitric acid, causticum, kali bichromicum* and *iodoform* may render good service.

EDEMATOUS LARYNGITIS.

Edema of the glottis or infiltration of the laryngeal mucosa with serum is an inflammatory condition of serious character. It may rarely follow the ordinary acute laryngitis or it may be a sequence of syphilitic or tuberculous invasion. The inflammation may come from severe diseases like diphtheria or it may extend from adjacent parts in such diseases as erysipelas of the neck and various forms of cellulitis. It is occasionally observed in such infectious diseases as scarlet fever, measles and typhoid. It may also occur as a complication in nephritis and heart disease, as they have a tendency to excite dropsical effusion. Enlargement of the cervical and mediastinal lymphatics, aneurysm of the arch of the aorta and thyroid overgrowth may develop the laryngeal edema by jugular compression. Caustics, hot liquids and foreign bodies may also cause it. It may likewise occur in angioneurotic edema.

Symptoms.—*Dyspnea* is the first and foremost symptom observed and in acute cases develops very rapidly. On account of the increasing obstruction the respiration has a marked stridor, but there is no evidence of cough. The voice usually becomes husky and there may be complete aphonia. The laryngoscope reveals an intense swelling of the laryngeal mucosa with a globe-like tumor of the epiglottis, and often this may be observed when the tongue is firmly depressed. The aryteno-epiglottidean folds are also a frequent seat of the edema and sometimes it exists below the true cords. The *diagnosis* is not difficult as the examination with or without the laryngoscope will reveal the edema; it may often be recognized by palpation with the finger. A fatal result may be expected unless there is prompt relief from astringents, ice, scarification, tracheotomy or intubation.

Treatment.—The *icebag* applied to the larynx and small bits of ice swallowed, often give prompt relief. Scarification of the swollen mucosa by means of a laryngeal lancet or even a curved bistoury may be demanded after the parts have been cocainized. Tracheotomy is demanded if relief is not obtained in this way. The local use of leeches is an ancient method of treatment. A solution of alum (grs. 20 to the ounce) or adrenalin applied with the cocain is often prompt in results. Among the internal remedies we should give first place to *apis mellifica.* This may be used in the second or third potency, or five grain doses of the first decimal potency have been advised as often as every two hours. *Cantharis, apocynum, arsenicum, kali bichromicum, pulsatilla* and *mercurius corr.* may be compared. Iodin and the iodids, such as the *iodid of lime, mercurius iodid* and *arsenicum iodid* are naturally indicated and should be used when indicated.

NEW GROWTHS IN THE LARYNX.

Neoplasms in the larynx occur as benign papillomas, fibromas, lipomas, cysts, myxomas, lymphomas, enchondromas and angiomas or as malignant sarcomas and carcinomas. Among the benign growths papillomas and fibromas are the most common. The papillomas develop on a broad base, are rarely pedunculated, and are inclined to recur. They are usually found on the vocal cords and their color varies from a light gray to a dark red. The tumors range in size from that of a millet seed to that of a filbert, or they may form in groups, giving a cauliflower appearance. The fibromas develop more slowly and are not liable to recur. They are located also on the vocal cords, are globular, nodular or pedunculated tumors and are usually pale in color. The malignant growths of the larynx are either carcinomas or sarcomas. The former is by far the most common, the epithelioma being the most frequently observed. The seat of the growth is generally on the vocal cords, the ventricle of Morgagni and the epiglottis.

Symptoms.—When the tumors are benign and very small there may be an absence of suggestive symptoms. When the neoplasms are large and develop rapidly, as in the case of malignant growths, a *marked hoarseness,* which increases to a complete aphonia, is the first recognizable symptom. When the tumor is located in the upper larynx a *cough* may succeed the hoarseness and aphonia. In case of obstruction *dyspnea* may be present. Should the epiglottis become involved there may be sudden orthopnea and asphyxiation. The *diagnosis* is confirmed only by laryngoscopic findings. The *prognosis* is favorable in the benign forms and very unfavorable in the malignant forms. The *treatment* requires the help of a specialist, as surgical measures alone succeed. In the malignant tumors little can be done toward a cure.

DISEASES OF THE BRONCHI.

BRONCHITIS.

Bronchitis is a catarrhal inflammation of the bronchial mucous membrane in whole or in part. It is divided into four varieties—acute bronchitis, capillary bronchitis, chronic bronchitis and plastic bronchitis. Capillary bronchitis, which involves the interstitial alveolar tissue is discussed as bronchopneumonia in the chapter on lung diseases.

Acute Bronchitis.—This is an acute catarrhal inflammation of the mucous membrane of the larger bronchial tubes. It is frequently called a "cold in the chest" and often involves the trachea, when it is more properly called tracheo-bronchitis.

Etiology.—The exciting causes are exposure to cold, climatic changes, variability of temperature, chemical, mechanical or biological irritants and extension of catarrhal inflammation from the naso-pharynx, larynx and trachea. Infectious diseases are also factors, measles, typhoid fever and influenza being the most common causes of this kind. Children and the aged are the most frequent sufferers. The predisposing causes are malnutrition, debilitating diseases, hereditary dyscrasiae and unhealthy environment. Enervating habits, occupations which exhaust the strength and too heavy clothing encourage it. Previous attacks, diseases of the lungs and heart which obstruct the circulation, and toxemic conditions are likewise predisposing causes. Tuberculosis should always be an etiological consideration.

Pathology.—The involved mucous membrane is hyperemic, swollen and its secretion is at first diminished. Later there is hypersecretion which changes in character according to the progress of the inflammation. The excess of mucus, the presence of degenerated epithelium and pus, together with a slight amount of blood, give a yellowish or greenish color. The submucosa becomes edematous and the tissues are infiltrated with leukocytes; the mucous glands are generally much swollen.

Symptoms.—The *onset* is marked by a mild chill or chilliness, sore throat, coryza and hoarseness, while convulsive symptoms may appear in delicate children. The *fever* is not high (101°-102° F.), and the pulse is increased in proportion. *Pain* and soreness, in varying degree, are felt in the back and limbs and in the beginning there is a sense of tightness and rawness, with a sensation of oppression in the chest. The *cough* is dry, irritating and paroxysmal, the expectoration is slight and viscid at first but becomes more abundant and muco-purulent later. As the purulent factor increases, the sputum is raised with greater ease and a general sense of relief is experienced.

Physical Signs.—Little is revealed by inspection, palpation or percussion. The *respiratory movements* are not generally increased except in cases with high fever and when the smaller tubes become involved; then dyspnea may be observed. Bronchial fremitus may sometimes be felt. The bronchial râles disclosed by auscultation are most characteristic, being at first dry, sonorous or sibilant but as the exudate becomes free, moist râles appear. The *character of the râles* depends upon the size of the tube involved; in the larger tubes they are coarse and in the smaller fine or subcrepitant. The râles often disappear temporarily after coughing.

Diagnosis.—The diagnosis of simple acute bronchitis is not generally difficult when the above symptoms are taken into consideration. The acute course, the slight fever, the dry cough, followed by the characteristic expectoration, the dry and moist râles, heard generally on both sides, and the absence of pulmonary consolidation together with the milder course of the disease should differentiate it from *pneumonia.* Should *broncho-pneumonia* develop during an attack of bronchitis the general condition becomes more serious, the fever increases, dyspnea and cyanosis develop. Auscultation discloses moist râles and broncho-vesicular breathing, while percussion will show small areas of consolidation. The unilateral apex consolidation, the temperature range, the cachexia and the sputum examination should easily define *pulmonary tuberculosis. Whooping cough* is often confusing until the characteristic "whoop" is recognized. The same difficulty exists in defining the cough which precedes the *eruption of measles.* These differentiations, however, are only a matter of careful study as the bronchitis progresses.

Prognosis.—The prognosis is generally favorable, as this is a disease which yields to prompt and careful treatment. The cough may continue some time but the extreme symptoms usually respond promptly to carefully selected remedies. The aged and the debilitated do not react so quickly. Inflammatory extension to the finer tubes is an unfavorable condition, while faulty elimination greatly retards the cure.

Treatment.—The *general care* of these cases should be most thorough until the acute symptoms subside. The patient should be put to bed, no matter how light the attack. The first attempt should be to relieve the irritation of the inflamed membrane and encourage secretion. For this purpose the hot compress, thoroughly covered with a woolen cloth, may answer in mild cases. The old-fashioned flaxseed poultice may suffice, but it is generally unpleasant and does not retain warmth. A mustard poultice, with a little oil added, is possibly a good application. The modern antiphlogistic plastic dressings are especially serviceable. Frequently a simple cotton jacket will suffice. The general tendency at the present date is to do away with any local applications. The room should be kept warm and at an equable temperature. Inhalations of steam containing the compound tincture of benzoin (dram 1 to the pint of boiling water) and medicated vapors are valuable adjuvants. The diet should be a simple one and the bowels should be relieved by enema or cathartic if necessary in order to encourage free elimination.

The best results are obtained by the use of *internal medication* in accordance with the predominating symptoms. In the early stages before the inflammatory process is well established, *aconite* is of greatest service; the exposure to cold air, the fever, thirst, restlessness, dry, hot skin, as well as the short, constant and irritable cough, are characteristic symptoms. It is generally claimed that this remedy should be used only for a short time, though my experience warrants its continued use. *Bryonia* may be considered when the characteristic sharp pain exists, as it has a decided affinity for the larger tubes and pertains to the earlier stages when the exudate is forming, the cough is dry and painful and the respiration is labored, an aggravation being produced by motion and the respiratory effort. The cough of bryonia is generally a dry one, with much constriction of the chest, fever not being excessive, though thirst is pronounced. *Cantharis* should always be considered in connection with bryonia, as it represents the extreme symptoms of that remedy with particular reference to the stitching pains. *Phosphorus* is indicated when the cough is dry, spasmodic and aggravated by entering a warm room; a sense of constriction within the chest is also a prominent symptom. *Ferrum phos.* is frequently demanded in children and plethoric people when the sputum is blood stained, while nervous symptoms may be marked. For the tendency to paroxysmal and irritating cough we can not find a better remedy that *hyoscyamus*. *Belladonna, sticta, gelsemium, kali bromatum, codein, rumex* and *sanguinaria* may also be considered here. The cough of *drosera* simulates the spasmodic dry cough of pertussis, with nausea and vomiting and marked contortion of the face during the cough are characteristic symptoms. *Pulsatilla* refers to the violent cough in which the mucus is tenacious and profuse, and is of great service in the bronchitis of measles. *Lachesis* also refers to a laryngeal irritation, attended by a dry and hacking paroxysmal cough, with aggravation from any external pressure.

As the acute inflammation begins to subside and the secretions increase, we naturally look to such remedies as *kali bichromicum, sulphur, hepar sulphur* and *antimonium tartrate.* The *iodid of antimony* is called for more when the expectoration is muco-purulent in character, while *antimonium ars.* may also be indicated. *Ipecac* is a valuable remedy with children. *Apomorphia* has a symptomatology similar to ipecac, though it is more applicable to middle-aged patients. *Stannum* is called for in subacute cases and refers to a profuse muco-purulent excretion which is easily raised, a sense of marked weakness in the chest being a prominent symptom. *Stannum iodid* should be compared. *Senega* should be considered in many of these cases and is always useful in the aged when the bronchial weakness is pronounced and much soreness is in evidence. The mucus is tough and tenacious, the cough is dry and persistent and there is much hoarseness. The majority of these remedies should be used in the third decimal potency.

CHRONIC BRONCHITIS.

This disease may be characterized as a chronic affection of the mucous membrane of the bronchial tubes. The inflammatory invasion may involve the entire bronchial structure.

Etiology.—It may be the result of repeated attacks of the acute form or be associated with such diseases as phthisis, emphysema, asthma, chronic alcoholism, rheumatism, gout, syphilis or cardiac and renal diseases. It is of frequent occurrence in the aged and is observed more frequently in climates subject to sudden changes. Exposure is the most frequent cause of the primary form. The inhalation of irritants produces it and the spring and winter seasons always furnish the majority of cases.

Pathology.—The mucous membrane is congested, thickened, grayish in color and the mucous glands are hypertrophied; again it may be atrophied to such a degree as to cause the submucous and muscular coats to stand out prominently. In the aged the cartilaginous structure suffers calcareous degeneration. The tube wall, in prolonged cases, gradually weakens and the air vesicles are also liable to dilate; thus there may be a resultant bronchiectasis or emphysema; dilatation of the right heart may also take place on account of this pathology.

There are really three varieties of chronic bronchitis which are differentiated by the character of the sputum. In the *dry form* the mucus is scanty, tenacious and raised only by violent coughing spells which favor the development of emphysema. The *moist form* is recognized by the copious expectoration, which may be mucopurulent or purulent; emphysema in this form is not so common. The *putrid form* is usually associated with bronchiectasis and gives a fetid odor to the patient's breath and to the sputum; the expectoration is mucopurulent and copious.

Symptoms.—The cough is the most characteristic symptom; it is severe in the dry form and usually paroxysmal. It is not so severe in the moist form, the paroxysms occurring in the morning to remove the excessive amount of exudate which forms during the night. It is aggravated in the winter and during unfavorable weather. It often disappears entirely during the summer. Fever is not marked; if it is present at all it is due to pus retention in the bronchial tubes and is observed mostly during the afternoon and evening. Pain is not very disturbing; exhaustion takes its place. Dyspnea is quite a feature and the patient often complains of being "short of breath," particularly when emphysematous complications exist. Asthma is sometimes a complication. Bronchorrhea, a profuse bronchial secretion when the aged are affected, is characterized by a frothy mucopurulent sputum; this is raised in great quantities in the morning.

Physical Signs.—*Inspection* shows a decrease in expansile power and some thoracic enlargement when emphysema is present; in such cases dyspnea is evident. *Palpation* yields a bronchial fremitus in cases of excessive secretion. *Percussion* reveals no abnormality except occasional hyper-resonance. *Auscultation* recognizes various rhonchi and a prolonged expiration. The respiratory sound is feeble. Moist râles are abundant and heard mostly at the base where secretion is excessive. Dry râles may be present at various stages of the disease, but they are not in evidence so much as the moist ones. Dilatation of the right heart may occur because of stasis in the pulmonary circulation.

Diagnosis.—It is an easy matter to recognize this disease when the typical symptoms are present. As chronic bronchitis is liable to furnish a favorable soil for the tubercle bacilli, it is best always to exclude *tuberculosis* in making a diagnosis. This is most reliably settled by microscopic examination of the sputum. The physical signs do not display the localized consolidations, the temperature range and the physical debility attendant upon pulmonary tuberculosis is either absent or much less than in the latter disease. *Fetid bronchitis* sometimes suggests *abscess or gangrene* of the lung. In *abscess* of the lungs, however, the chills, the septic temperature, the sputum containing pus and debris with elastic fibres should settle the diagnosis. In *gangrene* of the lungs the breath and the expectorated matter have a more intense fetor. The microscope reveals numerous elastic fibres, bacteria, mucopus and granular matter; the elastic fibres may be absent, due to a destructive ferment recently discovered in gangrene. *Bronchiectasis* is defined by physical signs which relate to a unilateral and limited involvement and the character of the sputum differs.

Prognosis.—When the disease occurs in the young or the middle aged the prognosis is not unfavorable. In the aged or infirm, it is a question if they ever fully recover from it. It is so frequently complicated with emphysema and cardiac or renal diseases that unfavorable symptoms are liable to develop at any time. It is the old person's cough which, though it may subside in the summer, is liable to recur in the fall or winter. Therefore, the prognosis as to a cure should be decidedly guarded.

Treatment.—All clinicians admit that the best treatment for chronic bronchitis is a change of residence to a warm climate, such as belongs to California, Colorado, New Mexico, Georgia, the Carolinas or other southern locations. An equable climate is most desirable. If this is impossible, the patient should be taught the efficacy of hygienic surroundings. Every locality may be improved in cleanliness, and advantage may be taken of the clement weather to live an outdoor life. Proper clothing must be used to insure warmth, and for this purpose wool underwear should be worn. Patients should be instructed how to develop and maintain a sufficient physical resistance by personal and general hygienic measures. The cool sponge bath daily, the open window day and night are essential in treating any case of this kind. The depreciated system is subject to recurrent colds which are always favorable to the aggravation of chronic bronchitis. The *diet* should be supporting if not stimulating. *Alcoholic stimulants* may be indicated for old people. The complicating diseases should receive thorough attention and especially the kidneys and heart should be carefully watched.

The relief of the irritating cough may sometimes be a mandatory requirement. For this purpose a balsam preparation may be indicated. A balsam composed of gum benzoin (ounce 1 1/2), balsam peru (ounce 1), aloes (grs. 15), alcohol (pint 1) is very efficacious. Two drams of this should be added to six ounces of glycerin. From this, teaspoonful doses may be taken occasionally.

Heroin and terpin hydrate will also be a useful adjuvant. *Codein* (gr. 1/8-1/25) frequently subdues the aggravating cough at night. These are suggested only as aids in the cases which demand relief when our indicated remedies fail to act. The *oil inhalations* give temporary help; eucalyptus or pine needle oil, combined with a slight amount of iodin, is probably one of the best preparations; carbolic acid may be added in the fetid forms.

The patient is often able to expectorate more thoroughly and relieve the distended bronchi by coughing "down hill"; with the head hanging over the edge of the bed it is possible to raise a great amount of tenacious sputum with ease. Some help is also obtained by compression of the thorax during the paroxysms of coughing.

When we consider the remedial agents, we should first think of the iodins. *Iodid of arsenic* is of great value when the discharge is profuse, irritating, and mucopurulent; there is marked general and cardiac asthenia. It should be continued for some time in aged patients—*iodin* will give better results when the membrane is very dry and the cough is irritable and frequent; it is always suggested when the emaciation is pronounced or there are any symptoms of tuberculous invasion. *Iodid of antimony* should be considered when the expectoration is mucopurulent; *stannum iodid* may be used when there is evidence of bronchial dilatation and much exhaustion after protracted coughing spells; the expectoration is thick, profuse, sweetish and easily raised. *Senega* may be compared to this but there is little expectoration, much rattling in the chest, bronchiectasis is often present, and gastrointestinal disturbances are common. While *senega* will give better results in five drop doses of the tincture, the *iodid of tin* should be used in the second or third decimal potency. *Ammonium carbonate* is a remedy which acts well in the bronchitis of the aged. The expectoration is difficult and there is respiratory failure and cardiac weakness; the mucus remains in the bronchi and is recognized by the large coarse râles. Rachitic or tuberculous patients with similar symptoms will respond better to *ammonium iodid. Sulphur* pertains to the tuberculous diathesis and a general glandular inefficiency, with signs of malnutrition. In this connection we should consider *silicea. Kali iodid* should never be overlooked when there is any syphilitic history. *Kali bichromicum* may be used in the dry bronchial catarrh when the cough is persistent and the expectoration is thick and ropy. *Tartar emetic* should be considered in this connection when the râles are abundant, but the expectoration is slight; usually the smaller tubes are involved; *bromin* aids mostly when there is laryngeal involvement and the exudate is croupous. *Spongia* is useful in the hoarse croupy coughs with slight expectoration. *Phosphorus* is always a good remedy when the sensation of tightness in the chest exists and there is hoarseness and aphonia.

Drosera is more frequently called for in chronic bronchitis than any other remedy; the spasmodic dry cough with paroxysms repeated so frequently that the patient can hardly breathe, the deep, hoarse voice, the aggravation at night while lying down, the oppression of the chest and the copious yellow expectoration are its leading symptoms. *Sanguinaria* pertains to the dry hacking cough which is followed later by expectoration of tough and stringy sputum; the cough is irritable in the beginning and is attended with dyspnea; later the expectoration is copious and offensive. Fever is also a feature of this remedy and simulates that of tuberculosis. *Causticum,* in some cases complicated by nervous debility, relieves the irritable cough when sphincter weakness exists and there is some tendency to laryngeal disturbance. This remedy has not a wide sphere of action and is only occasionally indicated. *Apomorphia,* in the third decimal potency, frequently helps the dry irritating cough attended by a sense of nausea. *Atropin* relieves the spasmodic irritating dry cough at night when the respiration is wheezing and difficult. *Rumex,* and *hepar sulphur, arsenicum, hyoscyamus, ipecac, grindelia robusta, chelidonium* and *ammonium mur.* should also be studied.

Calcarea carb., though not often thought of in relation to bronchitis, is nevertheless a remedy which has a decided sphere of action in cases where malnutrition is a factor. The strumous diathesis predisposes to bronchial weakness, and when the signs of lowered vitality are present we should think of *calcarea.* The tendency to recurrent colds, the constant disorders of digestion, the coldness of the hands and feet and the unhealthy skin are cardinal indications. The irritable cough at night, the disturbance of respiration from the slightest exertion and the copious and thick sputum call for its use.

BRONCHIECTASIS.

This is a circumscribed or general dilation of one or more of the bronchial tubes.

Etiology.—Unless congenital, which is rare, bronchiectasis is always secondary to other respiratory diseases. Lesions which weaken the bronchial wall, coughing and the accumulation of secretions cause the dilatation. This may be induced by any inflammation which leads to atrophic changes. Chronic bronchitis, emphysema and phthisis are frequently the contributing causes. In children it may follow measles, whooping cough and broncho-pneumonia. Obstruction by foreign bodies and tumors, aneurysmal compression or the contraction of lung tissue, incident to interstitial pneumonia, may be predisposing factors. The disease is more common to adult life.

The dilatation may be cylindric or saccular, and may affect one or more of the bronchial tubes. The *cylindric* form involves the entire length of the tube; it occurs more frequently in the medium sized and the smaller tubes, though the larger ones suffer a greater amount of thickening of the bronchial walls. The mucous membrane is thick and covered with catarrhal exudate. The *saccular* dilatations, varying in size, are generally multiple and are distributed along the course of a bronchus, the wall of which is atrophied and the mucous membrane is thin and smooth. The retained secretions, reinforced by the products of tissue degeneration, particularly in the large sacculated forms, are very offensive. Often the mucous membrane is ulcerated in spots, thus offering a seat for further inflammatory invasion. Tubercle bacilli may find such soil favorable to their development. The saccular variety may involve only a portion of the lung. Both the cylindrical and saccular forms may exist in the same lung.

Symptoms.—The characteristic symptoms are the violent cough and the copious expectoration of very offensive sputum which may amount to fifteen or twenty ounces daily. This expectorated matter is of a grayish-brown color and separates into three layers in a glass. A brownish froth appears on top, a thin mucoid layer comes next and the bottom layer is composed of a thick granular substance. Pus corpuscles, bacteria and, in case of much ulceration, elastic fibres are observed microsopically. The cough comes periodically when the dilated portion is distended with accumulated secretion, and is paroxysmal and severe until the sac is relieved. It generally occurs in the morning and for the rest of the day the patient is comparatively comfortable unless there is some other complicating inflammatory invasion.

The symptoms outside of this characteristic cough and expectoration, pertain more to the complicating disease which acts as a cause. As the bronchial dilatation supervenes, these symptoms become intensified. Dyspnea and pain may be present and if the ulceration is pronounced, there may be some fever which is usually characteristically absent. Hemoptysis, while a possible complication, is of rare occurrence.

Physical Signs.—These, to a great degree, are dependent upon the associate condition. The chest may have the barrel-shape of emphysema, or be retracted when chronic pleurisy or interstitial pneumonia are underlying factors; the normal mobility is diminished in either case. *Vocal fremitus* is increased in large sacculations which freely communicate with the bronchus. The *percussion* sound is flat, except in emphysematous conditions, or in case there are numerous saccules, when it becomes hyper-resonant. *Auscultation* reveals various moist râles dependent upon the character of the cavity. Bronchial breathing and occasionally râles of metallic quality are characteristic. Bronchophony and pectoriloquy are at times recognized. All of these features are usually located at the base of the lung. Circumscribed dulness which gives place to tympanitic resonance after coughing and expectorating, is a distinctive sign (Babcock).

Diagnosis.—A simple dilatation does not not always yield appreciable signs. Localized broncho-vesicular breathing, with occasional metallic râles, may be the only indications. The most characteristic sign is the sudden and periodical expectoration of a quantity of fetid, muco-purulent matter which arranges itself in layers, as described before. Then we notice the physical signs of a cavity which gradually disappear as the cavity refills. It must be differentiated from *fetid bronchitis* and *pulmonary tuberculosis*. *Fetid bronchitis* has the marked symptoms of a typical bronchitis which has assumed a chronic or subacute form; it is not so prone to emphysematous characteristics, nor is the expulsion of sputum so periodic. The expectoration is especially fetid and offensive. *Pulmonary phthisis* has a gradually increasing cough, fever, the expectoration is more persistent and varied in character, hemoptysis is frequently in evidence, the cough is not so periodic or paroxysmal, the sputum contains the tubercle bacilli and the progressive emaciation and cachectic conditions are marked. The tuberculous cavities are usually found at the apex, while those of bronchiectasis are usually observed at the base. The dulness in phthisis precedes the formation of a cavity, while in bronchiectasis the cavity is first in evidence. In *pulmonary gangrene* the systemic symptoms are grave and develop more rapidly, the sputum contains shreds of pulmonary tissue, and there is generally a history of pneumonia. *Circumscribed empyema* may simulate bronchiectasis, but it has also a history of a preceding pneumonia and the pus is the chief constituent of the exudate.

Prognosis.—Recovery depends upon the complicating factors and the degree of dilatation. The moderate dilatations in children or young adults, when secondary to diseases like measles and whooping-cough, are prone to recovery. The ordinary case runs a protracted course, the patient living for years, and is liable to remain in a chronic condition and finally die from some intercurrent disease or asthenia.

Treatment.—The chief object in treatment should be to correct and cure the complicating disease, and to remove the offensive sputum from the dilated tube. Whatever inflammatory invasion attends this disease should receive prompt and systematic remedial attention. The dilatation of the bronchial tube will never be helped until we remove the cause. Expectoration is aided if the patient, at times, coughs "down hill;" as an illustration, let him during a coughing paroxysm lie on a couch with the head and shoulders hanging over the edge; in this way it is often possible to empty the sac without the terrible coughing spasm which they frequently experience.

The use of antiseptic inhalations and injections have been recommended by many as a relief for the fetor and possibly as an aid in the curative process. *Carbolic acid, creosote, guaiacol, quaiacol-vasogen* and *potassium permanganate* are most frequently used in the inhalations and sprays. There are many devices in the market for this purpose, so it is not necessary to resort to home-made substitutes. Intra-tracheal injections were recommended by Stewart; he suggested a preparation consisting of menthol 10 parts, guaiacol 2 parts, olive oil 88 parts; a dram of this is injected twice daily, by a specially designed syringe, into the trachea. The difficulty experienced in using this correctly and the uncertainty as to the curative relief, to say nothing of the undue irritation if it is improperly introduced, would naturally condemn this application.

The use of the *internal remedy,* if persistently employed, will give us the best possible results. To be sure, the correction of the dilatation depends greatly upon the removal of the primary cause; still we must relieve the bronchial inflammation and overcome the constant irritation induced by the presence of the fetid exudate; therefore, any remedy mentioned under the treatment of bronchitis which may be indicated for this purpose should be administered. There are some remedies of unusual usefulness in this condition. The first I should mention is *terebinth;* this, in its pronounced action upon mucous surfaces, tends to facilitate the expectoration as it increases the amount of exudate rapidly, it stimulates the severe paroxysmal cough which expells it; the later action of this remedy aids greatly the reparative process in the bronchial mucous membrane. The symptoms which demand it are the rapid and difficult respiration, with an extreme oppressive, stuffy sensation in the chest, and the expectoration is copious and streaked with blood. *Terebene,* in five to ten minim capsules, has been used by some as often as every three or four hours in extreme cases. *Terebinth* should be used in the first decimal potency and repeated often, if the stomach will tolerate it. *Creosote,* in increasing doses, has been a favorite prescription with many practicians, but this is liable to irritate the stomach.

Stannum iodid is probably nearer in symptomatology to the conditions of bronchiectasis than any other remedy; it relates to the profuse mucopurulent expectoration resulting from bronchial dilatation; it also has the dyspnea, the fatiguing paroxysmal cough and the thoracic depression incident to the disease. *Hepar sulphur, silicea* and *iodid of arsenic* are to be similarly considered. *Silicea* more particularly refers to the purulent, lumpy and tenacious expectoration, the presence of which causes much obstruction to respiration. *Carbolic acid,* in the third to the sixth decimal potency, *kali permanganate* in the third potency, *balsam peru, ammonium iodid* and *ammonium chlorid* should be remembered:

BRONCHIAL STENOSIS.

Bronchial stenosis is a narrowing of the bronchus by constriction or compression. Constriction may occur from cicatricial contractions following ulceration or from the swelling of the mucosa in the smaller tubes. The bronchial walls may become thickened from inflammatory exudates due to syphilis, tuberculosis or possibly other acute or chronic diseases. Foreign bodies or new growths within the bronchus are frequent causes. Stenosis by compression may result from aneurysm, tumors, enlarged glands, mediastinal and pulmonary abscesses and pleural effusions.

Pathology.—The secretions of the affected bronchus are retained and the tube beyond the point of constriction is distended. Secondary inflammatory changes may follow and if the stenosis is complete, collapse of the lung may occur.

Symptoms.—These depend upon the degree of stenosis and size of the tube involved. *Dyspnea* is pronounced and the lungs are not properly filled with air, though the accessory muscles of respiration act with undue energy. *Expiration* is labored and the lower part of the thorax is retracted with each forced inspiration. *Pleuritic complications* may appear and localized dulness may be found as the result of the lung retraction and secondary changes.

Physical Signs.—On inspection the affected side shows restricted respiratory movement. *Palpation* reveals diminished tactile fremitus. The *percussion note* is changed only in case of atelectasis when dulness is found. On *auscultation* the normal vesicular murmur is markedly diminished on account of the obstruction. Dry and moist râles are heard at the point of obstruction. When a small bronchus is affected, there may be no marked perversion of the normal physical signs because a compensating emphysema may result.

Diagnosis.–The various râles at the point of constriction, with the percussion signs furnish the best diagnostic evidence. The history of the case should decide as to causative thoracic diseases. *Laryngeal stenosis* may be differentiated by laryngoscopic examination.

Prognosis.—This is generally unfavorable as the disease is liable to be protracted. It is decidedly dependent upon the predisposing disease. When foreign bodies create the constriction there may be some hope of removing them by surgical measures.

Treatment.—Except from the consideration of the symptoms which refer to the predisposing cause, our only relief may be obtained through surgical measures. Any foreign body should be removed and the constriction should be dilated with bougies when it is feasible. Emetics have been used to forcibly dislodge a foreign obstruction when it has not been surrounded by adventitious tissue. The spasmodic constriction of the bronchus is sometimes relieved by remedies which quiet the nervous tension; *chloral, valerianate of zinc, valerianate of ammonia* and *hydrobromate of hyoscin* are best in this respect.

ASTHMA.

The term properly applies to bronchial asthma, which is sometimes defined as nervous asthma. It is characterized by paroxysms of dyspnea, resulting from spasmodic contractions of the circular muscular fibres of the smaller bronchial tubes. It is observed mostly during the act of expiration.

Etiology.—*Heredity,* a constitutional peculiarity or the susceptibility of the bronchial muscular fibres are predisposing factors in the creation of this disease. The *exciting factors* may be found in a variety of conditions. Acute bronchitis, which frequently precedes it, often furnishes the hyperemia, the turgescent mucosa and the catarrhal exudate which excites the spasmodic contractions of the bronchial muscles; hence the term bronchial asthma seems to be warranted. Inhalations of noxious vapors, the pollen of certain plants, or the dust of the street may cause sufficient irritation to produce it. Damp weather or sudden changes of temperature may favor it. Gastric or intestinal disturbances, nasal polypi or uric acid excess, may be the fundamental factors in its production. To uterine irritations we may sometimes attribute it, though the disease is more frequent in males. *Cardiac asthma* is that form associated with diseases of the heart. We speak also of *renal asthma* when there is insufficiency or structural involvement of the kidney. Emphysema, gout, rheumatism, syphilis, rickets or even anemia may be provocative of an asthmatic attack. *Toxemias* of various origins, irritations of the pneumogastric nerve from the pressure of tumors and certain skin diseases are considered among the probable etiological factors. *Autotoxemia* is coming to be recognized as having great influence in the production of this disease.

Notwithstanding all these enumerated causes the underlying and essential influence is probably a neurosis. We find it associated with neurasthenia and even with epilepsy or the psychoses. Intranasal defects are given a prominent place by some observers in the production of this disease.

Pathology.—No lesion of the lung has been discovered which would account for the asthmatic attack. The pulmonary changes are entirely secondary. Irritation of the motor branches of the vagus probably accounts for the spasmodic contraction of the circular fibres of the bronchial tubes. Hyperemia and turgescence of the bronchial mucous membrane are also probable factors in the creation of the obstructive condition which causes the asthmatic spasm. Hypertrophy of the circular muscular fibres and thickening of the bronchial structure naturally lead to a diminished caliber of the tubes. A secondary emphysema may be observed. Dilatation of the right heart is also a resultant complication.

Symptoms.–The *attacks* appear quite suddenly and are marked by intervals of relief. The dyspnea is distressing and rapidly increases in severity until an intermission occurs. The accessory muscles of respiration are called into use; both inspiration and expiration are interfered with, but expiration is most difficult and is greatly lengthened, being accompanied with blowing and wheezing. Usually the number of respirations is decreased. As the dyspnea increases the face is pale and then cyanotic, with an expression of anxiety. The diaphragm is depressed and the patient gasps for air. He is obliged to sit up in bed and with the head elevated and the shoulders fixed he struggles to use every respiratory muscle. The extremities are cool and a clammy perspiration covers the body. A cough with slight expectoration is generally an attending symptom. As the respiratory struggle disappears the expectoration increases. Dry râles are heard at first but later moist râles are in evidence, when tough balls of sputum (perles) are expectorated. These can be unrolled in water and are found to be spirals of mucus which have been molded in the finer tubes (Curschmann's spirals). Leyden's octahedral crystals are also found in the sputum and are composed of phosphoric acid with an organic base. At one time these were supposed to excite the spasms by irritation. These, however, are found in other diseases but they are more constant in asthma. Relief from the spasmodic breathing is finally experienced and, greatly exhausted, the patient may fall asleep. Repeated attacks develop sooner or later.

Physical Signs.—The chest is barrel-shaped on account of the inability to expel the retained air. Despite the violent muscular efforts, the *respirations* are diminished in frequency and the expansile power is limited. *Inspiration* is short and gasping, while expiration is prolonged. The *percussion note* is hyper-resonant if there is emphysematous development. *Auscultation* reveals a variety of râles which are interchangeably dry and moist and are heard best on expiration.

Diagnosis.—The history, the appearance of the chest, the character of the respiration and the harsh râles heard during expiration should make the diagnosis. In *laryngeal dyspnea* there is inspiratory and not expiratory difficulty and the chest is diminished in size. The *symptomatic dyspnea* which attends cardiac and renal diseases should be differentiated by the diagnostic symptoms of those diseases. In *emphysema* there are different causes; the dilatation of the air cells is attended with changes in lung structure, the dyspnea is not paroxysmal and the sputum has not the same characteristics.

Prognosis.—If it is possible to find the cause and remove it, recovery is probable. The disease is liable to develop chronic bronchitis, emphysema and dilatation of the right heart. The percentage of recoveries is not great, though there is little danger to life.

Treatment.—The principal effort in the treatment should be to remove the neurotic cause; this may be done only by a painstaking study of the patient. The next demand is to relieve the spasm; this is of the greatest importance to the patient and for this he calls the physician. Therefore, it is necessary to resort to every expedient which experience may offer for this purpose. *Potassium nitrate* is a standard adjuvant; a piece of blotting paper soaked in a saturated solution, dried, and burned in a saucer, will often give temporary relief by inhalation of its fumes. *Chloroform* or *ether* inhalations will sometimes shorten, if not overcome, the spasm. Often a few drops taken internally with a little water will act like magic in this respect but the effect is not generally lasting. *Amyl nitrite* also is a popular inhalation. Fumes of *stramonium, chlorate of potash* and *tobacco* have been recommended by some authors. In severe cases there is no better temporary measure than the hypodermatic use of *morphin* and *atropin* (gr. 1/4 and gr. 1/150); this should be repeated if necessary. The great objection to this is the danger of forming the morphin habit, as the patient will demand its use for every paroxysm. *Chloral hydrate,* in ten grain or larger doses, or *sodium bromid,* the same dose, will in many cases work nicely. The latter may be repeated every hour for three or four doses, or it may be given in larger doses at first. *Chloralamid* (gr. 10-20), *sulfonal* (gr. 10-15), or *trional* (gr. 10-25), may be efficacious in certain cases. Some have advised the use of black *coffee* in large amounts, while others commend the use of *whiskey,* even to intoxication. These expedients have not served me well. As a general thing we should avoid sedatives if possible.

Recently I have been informed by a trustworthy physician, that equal parts of *peroxid of hydrogen* and water, when given in teaspoonful doses half hourly or hourly, will relieve the spasm. *Adrenalin* has been recommended in doses of 3 to 6 minims of the 1 to 1000 solution. It will cut short the attack in from two to twenty minutes. *Hydrobromate of hyoscin* (gr. 1/200-1/100) every two or three hours relieves some cases when mental excitement is extreme. *Atropin,* in gradually increasing doses, has been commended and in some cases it acts better alone than when combined with morphin. Atropin (1/150-1/100) hypodermatically, will often relieve the acute spasm. All sources of reflex irritation should be removed.

The *diet* should be regulated in every case; the patient should not be permitted to eat heartily at night. The relief of uric acid excess is at present a consideration of importance and, therefore, the exclusion of meats may be a temporary or permanent necessity. Some have claimed decided results by forcing renal elimination through the use of salts and alkaline waters in abundance. In this respect we may increase the amount of urea eliminated by the use of *diuretin,* in ten grain doses, three times daily. *Bitartrate of potassium* may serve us equally well in this respect. I am confident that I have, by these measures relieved and to a certain extent aborted the approaching spasms when I have had sufficient warning. Inhalations of *oxygen* have been of some benefit. *Exercise,* the cool sponge bath, the well-ventilated room, the regulation of habits and, in fact, the systematic adoption of the rest treatment will greatly aid in preventing the attacks. Especially should those measures be enforced which will increase the elimination.

Of the remedies used internally during the paroxysmal state I prefer *grindelia robusta*; this has given me best results in five to ten drop doses of the tincture repeated at first every hour or two for a few doses and then less frequently. From the standpoint of symptomatology it represents the typical paroxysmal features of the disease; the patient feels drowsy, but is unable to sleep on account of the recurrence of asthmatic breathing; it also pertains to the reflex gastric irritation which so frequently excites the paroxysm. The continued use of the remedy in potency during the inter-paroxysmal state has apparently cured some cases. *Ipecac* I regard as next in importance; its prompt action upon the vagus disturbance tends to correct both the asthmatic condition and the gastric irritation which attends it; nausea and vomiting always are its characteristic symptoms; dyspnea and precordial distress and a wheezing, distressing cough are disturbing features. This remedy has served me well in the lower potencies. *Lobelia* has for a long time been a favorite remedy. It has been used in five to ten drop doses of the tincture, but its emetic action and its disturbance of the heart often necessitate smaller doses. Great prostration, nausea, vomiting and considerable chest constriction are its chief symptoms. *Apomorphia,* in the third decimal potency, will often control a severe spasm. *Aspidospermin* acts well when nausea and a loose but irritable cough are in evidence; in many respects it corresponds to *tartar emetic* which may be used in many chronic cases. *Moschus* has been praised for its action when hysteria is an underlying factor. The internal use of *amyl nitrite* and *glonoin* in the third potency is efficacious when the spasms assume the epileptoid feature. *Cuprum ars.,* is probably the best form of arsenic to use when the typical arsenicum symptoms are present; when this is indicated we generally observe epigastric and abdominal paroxysms as well. *Arsenicum iodid* should be considered here.

In the truly chronic or the inter-paroxysmal stages of asthma, we may consider several remedies which are available. Of these I would suggest a careful study of the *monobromid of camphor* in the first decimal potency; it relieves the nerve irritation and the neurasthenic excitement which precede or develop the asthmatic attack. *Camphor* in small doses is a nerve sedative not fully appreciated but in combination with the bromid it gives even better results. *Nux vomica* or *ignatia* will yield results when the symptoms of these remedies are present; they refer to the reflex excitement of the pneumogastric as the result of gastric disturbances. Sedentary individuals, those suffering with gastric perversions from alcoholic excess, and hysterical patients will respond to these remedies. *Sulphur* is indicated when the skin diseases have been suppressed, or when the rheumatic or other diatheses antedate the symptoms of asthma; it will give best results when used in the potency. *Arsenicum* is serviceable when the attacks occur after midnight and when the exhaustion, anemia and the general symptoms of this remedy are present. The *arsenite of antimony* or *strychnia ars.,* prove more effiacious in certain cases. *Bryonia* is often indicated when we find a frequent recurrence of bronchial attacks in which sharp and lancinating pains are constant; the excessive thirst and the characteristic gastric pain are also present. *Yerba santa, yerba manza, phillandrium agnafolia, eucalyptus, ptelia* and *rumex crispus* should always be kept in mind when treating a case of asthma. Cases of asthma due to nasal disease will require surgical aid to remove the cause, which is usually an enlarged middle turbinate or polypi pressing on the septum.

FIBRINOUS BRONCHITIS.

This is also termed croupous, plastic or membranous bronchitis. It is an acute or chronic inflammation of the bronchial mucosa in which a fibrinous exudate forms within the bronchi and is ejected during severe coughing paroxysms. This appears as a cast of the bronchial tubes and their ramifications; casts are solid from the smaller bronchi and hollow from the larger ones.

Etiology.—This is a very rare disease and the cause is undetermined. Constitutional causes are evidently basic, for it is usually observed in the feeble, the anemic and in males. It occurs generally in young adults and is more in evidence during the early spring; it is also associated with tuberculous conditions. Antecedent skin affections such as herpes, impetigo and pemphigus are claimed by some authors as contributory influences. Streptococci have been found in the exudate. It may accompany the membranous form of laryngitis.

Pathology.—Our pathologic knowledge of this disease is not complete. The exudate has a grayish-white color; is attached quite firmly to the mucous membrane which it denudes of epithelium when it is expectorated; it appears as casts which correspond to the parts of the bronchi affected. It may involve only portions of the bronchial structure or it may be ejected in the form of a bronchial tree. The casts are composed of coagulated fibrin in concentric layers; included in these are found leukocytes, epithelium and mucus.

Symptoms.—In the *acute form* the appearance of *chills* and *fever* may initiate the attack. *Dyspnea* is marked, as the respiration is obstructed. These symptoms may be preceded for a time by those of acute bronchitis. Violent, frequent and paroxysmal *spells of coughing* appear early and are ultimately followed by expulsion of the bronchial casts. Temporary relief is experienced after this. *Hemoptysis* attends or precedes the expulsion of a cast, but it is rarely lasting or serious. Some amelioration of symptoms follows the hemorrhage. The condition may temporarily resemble that of catarrhal bronchitis with a slight mucopurulent expectoration. The sudden suppression of the cough, the absence of membranous expectoration, particularly with the reappearance of dyspnea, oppression and temperature, are grave signs which point to asphyxia. At times there is an absence of the positive signs of fibrinous exudate and the diagnosis is only confirmed at the autopsy. The acute cases do not run longer than a few days, or at most two weeks. The quantity of fibrinous exudate gradually disappears from the sputum, the temperature improves, the cough abates and the respiration approaches normal as the disease subsides.

In the *chronic form* the attacks occur at varying intervals and the severe symptoms are not present. It is usually secondary to protracted chronic bronchitis and may be associated with more severe lung complications. There are often remissions of various duration. The fever, dyspnea, and cough, in the chronic form, are not so pronounced. It may assume, however, all the typical symptoms outlined in the acute form.

Physical Signs.—The obstruction caused by the casts limits the amount of air entering the corresponding part of the lung; hence tactile fremitus and expansion over the affected area are diminished or absent. The normal respiratory murmur returns after the expulsion of the cast. Percussion resonance is diminished over the area involved. Both dry and moist râles appear at different times.

Diagnosis.—The examination of the casts will settle the diagnosis. When these appear in small particles it is somewhat difficult to decide; in water they may unfold and assume a tree-like shape. The presence of mucin or fibrin in the sputum will differentiate from ordinary bronchitis. In *diphtheria* we must rely on the microscope to distinguish by finding the Klebs-Löffler bacilli. We find also in diphtheria that the exudate begins on the pharynx and tonsils and extends downward to the bronchi usually.

Prognosis.—In the acute form the disease may terminate suddenly by death from asphyxiation or hemorrhage. If the obstruction persists the prognosis is decidedly unfavorable. Any constitutional complication makes the outlook worse. The chronic form is not so dangerous though it usually tends to recur during a long period of time.

Treatment.—The first aim, in the acute attack, should be to relieve the obstruction caused by the exudate; to facilitate this steam *inhalations* containing iodin, creosote or lime are most efficacious. *Sprays* of lactic acid or guaiacol-vasogen may be resorted to. *Dry heat* applied to the chest may help. *Emetics* should be promptly employed in severe cases in order to get rid of the obstructing cast and overcome impending asphyxia. The signs of cyanosis should always indicate the use of an emetic. *Pilocarpin* (gr. 1/12-1/6) hypodermatically should be resorted to in grave cases. *Strychnia* should always be used with this to obviate the dangerous cardiac depression. In the chronic forms much relief is obtained by a change of climate particularly to those of higher altitude.

The most serviceable remedies are those containing iodin. Therefore *iodin, calcarea iodid, iodid of arsenic, ferrum iodid, kali iodid* and *baryta iodid* should first be considered. The *iodid of lime* is without doubt the most valuable and should be used in heroic doses. *Bromin, spongia, acetic acid, picric acid* and *lactic acid* should next be thought of. *Silphium, stannum, hepar sulphur, tartar emetic* and *kali bichromicum* are sometimes indicated.

DISEASES OF THE LUNGS.

CIRCULATORY DISTURBANCES IN THE LUNGS.

Active Hyperemia.—This may take place following violent physical exercise, from extreme nervous excitement and as the result of alcoholic intoxication or the inhalation of hot air. It is likewise a symptom of such pulmonary affections as pneumonia, bronchitis, pleurisy, tuberculosis or anemia of the opposite lung. The *pathology* is characterized by marked injection of the blood vessels in the bronchial mucosa, prominent capillaries in the alveolar walls and swollen and granular alveolar epithelium. A blood stained frothy fluid is observed upon section.

Symptoms.—*Dyspnea* is the characteristic symptom inasmuch as rapid respiration is a compensating necessity to oxygenate the blood, which function is impaired owing to the diminished capacity of the air cells. A *cough,* with rusty and frothy expectoration, is another distinctive symptom. The *physical signs* comprise the bilateral involvement, the increased tactile fremitus, the impaired percussion note with imperfectly defined pitch, and the broncho-vesicular and occasional bronchial breathing. The *diagnosis* is made by the sudden appearance of the frothy and bloody expectoration which attends the dyspnea and cough. The absence of fever is also a distictive feature. The *prognosis* depends upon the duration and the subsequent disease of which this may be a symptom. Unless prompt relief is obtained a serious result may follow.

Treatment.—*Cardiac stimulants*—digitalis, nitroglycerin or strychnia—are demanded at once and it is best to administer these hypodermatically repeating the dose in two or three hours if necessary. Venesection is warranted in nearly every case and hot local applications are also to be employed. Leeches, turpentine stupes and cathartics are also adjuvant measures. *Aconite, veratrum viride, ferrum phos.,* and *belladonna* will render good service according to their indications.

Passive Hyperemia.—This always is either mechanical or hypostatic. *Mechanical hyperemia* is due to those conditions which impede the blood in its return to the left heart. This usually arises in case of mitral stenosis or regurgitation, dilatation of the right ventricle and cerebral diseases. The *pathology* is defined by the voluminous lungs, the distended pulmonary vessels and the presence in the air-spaces of the venous blood. The lung tissue has a reddish-brown color and as there is an increased resistance in cutting, the term "brown induration" has been employed. On section this color rapidly assumes more of a reddish hue as a result of the oxidation of the hemoglobin. The alveolar cells contain altered blood-pigment and the interstitial connective tissue is increased. The symptoms include the dyspnea, cough and frothy or bloody expectoration which result from the lung engorgement.

Hypostatic Hyperemia.—This is observed when the heart is weakened by infection, exhaustion or old age. It may also exist as the result of prolonged dorsal decubitus. Organic growths, and such infections as tuberculosis and rheumatic fever, may constitute the underlying cause. The affected parts of the lung are dark-colored and distended with a transudate of blood and serum. This condition gives rise to hypostatic pneumonia which is a mild form of lobular pneumonia.

The *symptoms* are not well defined and in many cases none are recognized. Cyanosis may be detected in extreme cases or in poorly nourished patients past fifty. Careful physical examination will reveal increased fremitus, slight dulness, impaired vesicular breathing and sometimes bronchial breathing with liquid bubbling râles. (Anders.) The *prognosis* depends largely upon the causative disease. The *treatment* must look first to the cause which induces the hyperemia. Prompt stimulating treatment of the impaired heart will give greatest relief. Strychnia, digitalis, strophanthus and other cardiac stimulants should be pushed to the point of physiological action and their hypodermatic use is often required. When there is intense pulmonary engorgement, bleeding should be practiced and twenty to thirty ounces of blood should be drawn from the vein. Change in the patient's position is also necessary, as the dorsal decubitus usually favors the hyperemia.

Pulmonary Edema.—Edema of the lungs is usually the result of an intense pulmonary congestion with a transudation of serum into the air-vesicles and interstitial tissue. Two forms are recognized. (1) *Collateral edema* is a circumscribed involvement of the lung which usually appears in connection with pneumonia, abscess or pulmonary infarction. (2) *General pulmonary edema* affects a greater portion of the lung, the base being mostly involved, and hydrothorax is often present. With the congestion the lung is dark in color and the serum is blood-tinged. The affected lung-tissue is heavier than normal, though it does not sink in water. It has a boggy feel, pitting is observed on pressure and on section a serous or sero-sanguinolent fluid exudes from the cut surface.

The *cause* of pulmonary edema is not well understood. It may be due to the increased tension in the pulmonary circulation as the result of cardiac weakness. Obstruction to the outflow, following left ventricular weakness or aortic stenosis, creates an increased arterial tension as well as a right heart paralysis and both of these favor pulmonary edema. Toxic and infectious diseases, together with blood changes, impair the nutrition of blood vessels and as a result the vessel-walls suffer an increased permeability which favors pulmonary edema. Pneumonia, valvular heart disease, anemia, nephritis, cerebral hemorrhages and acute infectious diseases alike predispose to heart weakness and are, therefore, etiological factors in the development of this diseased condition. Vasomotor relaxation is another cause which may favor this perversion.

The *symptoms* of edema of the lungs include first of all the dyspnea which is proportionate to the degree of involvement. A *cough* is usually attended by bronchorrhea and the sputum, which is expectorated with difficulty, is usually copious, frothy and blood-stained when congestion is a factor. The sputum is sometimes raised only by great effort, and may be so tenacious that laryngeal obstruction is imminent. In the inflammatory type fever may be pronounced, the pulse being accelerated and weak, the extremities are cold and cyanosis is a typical symptom. The *physical signs* will aid the investigation of the symptoms. Dulness is recognized over the affected area, broncho-vesicular breathing displaces the normal vesicular murmur and small liquid râles are audible on inspiration and at the beginning of expiration over the seat of involvement.

The *diagnosis* rests to a great extent upon the bilateral dulness at the base of each lung, the bubbling râles in this vicinity and the absence of fever unless the disease assumes an inflammatory type, or is associated with infections. *Hydrothorax* will be differentiated by the change in the upper level of dulness when the position of the patient is altered. Moist râles are also absent in hydrothorax. *Broncho-pneumonia* is accompanied by fever, the areas of dulness are more sharply defined and the sputum is glairy and tenacious. The *prognosis* depends upon the gravity of the causative disease and the promptness with which relief is obtained.

The *treatment* includes primarily the removal of the cause. If the cause happens to depend upon faulty cardiac action we must give special and continuous attention to this. The only way to care for a weak heart is to stimulate persistently with *strychnia* (gr. 1/60) or *digitalin* (gr. 1/100) hypodermatically every three or four hours with atropin, or other cardiac and respiratory stimulants until we are positive the heart can perform its work. Such active purgatives as elaterin, croton oil or calomel may give great relief by causing profuse watery stools. *Change in position* is a mandatory requirement, for by this procedure the blood can not gravitate to the dependent parts. We too frequently allow our pneumonia patients, as well as those suffering with other diseases, to lie on the back or assume one position for too long a time. Venesection, cupping, diuresis and diaphoresis are also essential features of the treatment. The homeopathic remedies mentioned under the treatment of pneumonia, cardiac and other diseases which favor edema should be used according to their indications.

Hemoptysis.—This term properly applies to a pulmonary or bronchial hemorrhage. It refers to the expectoration of blood in large or small quantities, which may come from the bronchial mucous membrane of the smaller tubes, or from the lung parenchyma. Hemorrhage from the mouth, posterior nares, larynx or trachea may be confused with true hemoptysis.

Etiology.—Pulmonary tuberculosis should always be suspected as a cause for it is the most common though not the only cause. When hemoptysis appears early in the disease, it probably arises from extreme congestion in the small bronchi; later it may occur from the erosion of a pulmonary blood vessel; still later, if a cavity be its source, it will be mixed with debris. Pulmonary congestion may give rise to slight hemoptysis, and this may be aided by a mitral lesion of the heart. Hemorrhagic infarction, croupous pneumonia, fibrinous bronchitis, carcinoma and gangrene of the lung may all contribute to hemorrhagic expectoration. Acute infection, aortic aneurysms, atheromatous blood vessels, gout and interstitial nephritis as well as hemophilia, purpura hemorrhagica and anemia may also be predisposing causes. Traumatism affecting the chest, paroxysmal coughing as observed in whooping cough, obstruction from emboli or tumors and aneurysm of the pulmonary artery may give rise to hemorrhage from the lungs. Vicarious menstruation may also assume the form of hemoptysis but is very rare.

Pathology.—In ordinary cases the hemorrhage is due to the rupture of the finer capillaries in the bronchial mucous membrane. The weakness and the over-distention of the capillary walls permit this in association with bronchitis and any cardiac disease. Destructive changes in the lung parenchyma may lead to the erosion of a larger vessel. We find this condition in cases of tuberculous destruction or ruptured aneurysm. The mucous membrane at autopsy presents a swollen and puffy appearance; occasionally it looks anemic from the continuous hemorrhage. A marked spot of hemorrhage is often detected or small ecchymotic areas are visible. The alveoli may be filled with blood or, in case of a cavity, a considerable amount of coagulum may cover the bleeding point.

Symptoms.—The *occurrence of hemorrhage* is suggested by few premonitory signs. The patient may experience a salty taste in the mouth and a cough may precede the expectoration of blood. In ordinary cases the blood appears only in the blood-stained sputum which follows the cough. In other cases, when the hemorrhage is extreme, there may be no cough and only the symptoms of shock—feeble pulse, subnormal temperature, pallor and prostration are prominent. Usually the patient is restless and depressed. More rarely fever and delirium may follow. *Slight hemorrhages* may take place from time to time when no structural change affects the arteries or the patient may experience only one severe hemorrhage when there is no organic cause. When *pulmonary tuberculosis* is present the hemorrhage is liable to be severe and will probably be repeated at intervals. Attending this we shall find the other symptoms of phthisis or they will rapidly develop after the hemorrhage, which may often be regarded as the premonitory sign. Usually the blood is bright red in color and very frothy. When there is a *large cavity* in the lung, the hemorrhage may be so severe as to cause death. The blood may be swallowed and vomited, thus leading to the suspicion of gastric ulcer, or it may pass into the intestine, giving a dark color to the stools.

Differential Signs.—In the *slight hemorrhages,* with little coughing, we may find the heart at fault, or there may be a persistent bleeding from an aneurysmal dilatation. When, however, the aneurysmal sac ruptures, there is generally a pronounced bleeding into the respiratory tract which is suddenly fatal. *Ulceration* in the bronchial mucosa will give slight hemorrhages, but in cases of emphysema or chronic bronchitis there may be occasional coagula with the signs of casts. *Traumatism* may cause trivial hemorrhages which may be accounted for by chest injuries and vicarious bleeding has been mentioned in cases of perverted menstruation. Generally we shall detect moist râles in the larger bronchi or marked signs of the destruction of lung parenchyma. When *pulmonary tuberculosis* is the cause the progressive symptoms of that disease will confirm that physical condition. *Epistaxis* may confuse us when the blood enters the naso-pharynx and causes a cough to expel it. An examination of the posterior nares will settle this. *Laryngeal hemorrhage* is not excessive and is mixed with much mucus. *Hemorrhages from the mouth* ought also to be recognized by local examination. In *hematemesis* the blood is coagulated, dark in color and acid in reaction; it is expelled by vomiting. Prune-juice sputum generally characterizes *pulmonary cancer.* In *pulmonary hemorrhage* we shall usually find the aerated and clear blood which is alkaline in reaction; it is invariably thrown from the mouth by the act of coughing. In addition to all of these differentiating points a careful physical examination will reveal the pulmonary involvement.

Prognosis.—As hemoptysis is so often a forerunner of phthisis, we should invariably look upon it as an unfavorable symptom. It may be possible to stop the bleeding in mild cases, but subsequently the graver pulmonary features may appear.

Treatment.—The treatment will depend greatly upon the physical condition of the patient, the extent of the hemorrhage and the cause. In all cases absolute quiet must be preserved and the patient must be kept in bed. The food should be of the simplest character and no stimulating or hot drinks should be permitted. Expectoration should be aided by the position of the patient and every local and specific agent within our reach. Morphin hypodermatically may be necessary for its sedative action. The temperature of the room should be low, the extremities should be kept warm and yet the patient should not be burdened by excessive bed-clothing. Cool drinks and bits of ice should be administered by the mouth frequently. The icebag should be applied to the chest in the majority of cases. Cardiac sedatives should always be employed to prevent any undue pulmonary arterial pressure. *Aconite* will be our first indicated remedy on account of the nervous excitement and for the sake of the arterial sedation. The coagulability of the blood may be increased by *nitric acid, gallic acid* and the *acetate of lead. Calcium chlorid* (grs. 5-20) in solution may also be given four times daily to increase the coagulability of the blood. *Hamamelis, ipecac, arnica, phosphorus* and *ferrum phos.* are worthy of consideration at all times. *China, geranium, trillium, sabina, ipecac* and *millefolium* will be called for according to indications. *Erigeron* and *secale* should also be thought of. *Hydrastin hydrochlorate* is one of the best remedies for long continued use and such remedies as *digitalis, convallaria, crategus* and *adonis vernalis* will be called into use when the heart is at fault.

Pneumorrhagia.—Pulmonary apoplexy exists when hemorrhage takes place into the air cells and the lung tissue. This may or may not be attended by injury of the lung parenchyma. When the parenchyma is lacerated the hemorrhage will be diffuse, but when there is no parenchymal rupture the effused blood will be circumscribed within the air cells and the interstitial tissue. Diffuse pulmonary apoplexy follows the rupture of a thoracic aneurysm which is attached to the lung surface. Penetrating thoracic wounds are most frequently the cause of pneumorrhagia.

Symptoms.—The symptoms of pulmonary apoplexy are not well defined. The diffuse form may give expression to a profuse hemoptysis, pronounced dyspnea, cyanosis and collapse. The *physical signs* will give evidence of sudden and extensive lung consolidation. The circumscribed form will be recognized with difficulty. The prognosis is unfavorable, as gangrene and abscess frequently result, should death not occur as the result of the hemorrhage.

Treatment.—Treatment does not offer much encouragement. The patient should be kept absolutely quiet in bed, as the object is to encourage the formation of a clot if possible. *Cold applications* to the chest and bits of ice in the mouth offer some relief in arresting the hemorrhage. Ergot should not be employed, though it is possible to obtain some help from such remedies as *secale, ipecac, hamamelis* and *hydrastin hydrochlorate,* in the second or third potencies. The cardiac stimulants should be employed when demanded, but at best a fatal termination is to be looked for.

Pulmonary Embolism.—This occurs as the result of an embolic or thrombotic obstruction of the pulmonary arteries. Hemorrhagic infarction takes place most frequently as the result of stasis when the pulmonary circulation is feeble. Diseases of the lungs and heart are the most common predisposing causes, thrombi in the right heart and the systemic veins often being the source of the emboli. Infectious emboli may give rise to pulmonary abscesses and hemorrhage infarction.

Pathology.—"Embolic infarctions are firm, airless, brown or black, wedged-shaped masses, with their bases usually at the pleura, which soon becomes lustreless and covered with a delicate layer of fibrin." They may be single or multiple, usually small in size, and are located most frequently at the back of the lower lobe though, in rare instances, a greater portion of the lung may be involved. Leukocytes and red blood-corpuscles are found in the air cells and the alveolar septa. Edema and collateral congestion are often present and rarely a pneumonic consolidation is in evidence.

Symptoms.—*Dyspnea* appearing suddenly, the patient making frantic efforts to get air, is the most characteristic symptom. *Hemoptysis* is the next important symptom and is usually present in case of mitral disease. A *cough* attended by chest pain then follows, in which the patient expectorates a dark, gelatinous and mucoid sputum. Many times death occurs before any suggestive symptoms develop. This is always true when occlusion of a large branch of the pulmonary artery occurs. In other instances death may quickly follow the appearance of extreme dyspnea, syncope, convulsions and loss of consciousness. The *physical signs* in the case of a well defined consolidation following the embolism consist in increased fremitus, dulness, moist râles and bronchial breathing. These signs are often negative in case of small and deep seated infarctions and the physical symptoms are always liable to be influenced by the presence of bronchitis, edema and collateral consolidation. A friction sound will always favor the diagnosis. Fever may or may not be present and cardiac enfeeblement with all its train of symptoms is liable to be present in every case.

Diagnosis and Prognosis.—The disease is diagnosed by the etiological history and the sudden appearance of dyspnea, cough, bloody expectoration, chest pain, the loss of consciousness, convulsions and the physical findings which reveal a well defined consolidated spot in the lung. The *prognosis* is usually grave though the absorption of the embolism is not impossible in which case the symptoms gradually subside. Gangrene and abscess are possibilities in the case of bacterial invasions. A fibroid change, which leads to contraction and occasional calcification, sometimes ensues.

CHRONIC INTERSTITIAL PNEUMONIA.

This is a chronic disease of the lungs in which there is an increase of connective or fibrous tissue. It is also termed fibroid induration and cirrhosis of the lung. It occurs both as a primary and as a secondary disease, the latter being more frequently met with.

Etiology and Pathology.—The disease is rarely primary and generally follows prolonged inflammatory conditions of the lungs. Pulmonary tuberculosis, emphysema and adhesive pleuritis predispose to it. Delayed resolution following lobar pneumonia is an exciting cause; the exudate which fills the air cells is organized into connective tissue. Atelectasis from compression may often be an associate cause. Induration extending into the lung tissue as a result of broncho-pneumonia is a most frequent cause. Tuberculosis in its various degenerative phases may predispose to it and the same may be said of the fibrinous bands resulting from adherent pleural membranes.

Symptoms.—Cough which is gradually aggravated as the disease progresses is a constant sign. There may be a mucous or sero-mucous expectoration. Dyspnea is a persistent symptom and pain may attend the fibroid development. General emaciation and asthenia are also present.

Physical Signs.—The affected side of the chest is retracted while a compensating emphysema is noticed on the opposite side. The side involved is fixed during respiration and the heart is drawn toward the affected side. When the right side is implicated the apex of the heart will be drawn beyond the right of the sternum and when the left lung is affected the apex will be displaced to the left. The ribs are approximated, the intercostal spaces are obliterated, the chest is shrunken and the shoulder droops, all of these symptoms resulting from the fibrous contraction.

By *palpation* the loss of expansile power and increased tactile fremitus may be recognized. *Percussion* generally reveals the dull note of consolidation, while *auscultation* reveals the bronchial breathing which may become sonorous or cavernous, and various dry and moist râles may be heard.

Prognosis and Treatment.—The disease generally runs a chronic and uneventful course. Death is hastened by this disease, usually resulting from some intercurrent complication such as pneumonia or from valvular insufficiency and cardiac dilatation. The *treatment* applies simply to maintaining the general health and to the relief of any acute disease.

BRONCHO-PNEUMONIA.

This is a catarrhal inflammation of the terminal bronchi and the air-vesicles which form a pulmonary lobule, hence the interchangeable terms broncho-pneumonia and capillary bronchitis. It is an infectious disease and should be classified under that head, but from custom it is included among the pulmonary diseases, inasmuch as a specific organism has not yet been discovered.

Etiology.—It most frequently affects young children and the aged, being generally secondary to other diseases. It develops by extension from simple acute bronchitis, though the inflammation may start in the finer tubes. Ordinarily it is bilateral and affects a large number of the smaller tubes. Previous attacks of bronchitis, debility, alcoholism in the adult, and cachectic states predispose to it. Infectious diseases and rickets are common factors which favor it in children. It may also develop as a complication in typhoid, smallpox, erysipelas and influenza, seriously affecting the prognosis.

Pathology.—The lungs in broncho-pneumonia appear mottled, due to the areas of atelectasis, consolidation and emphysema; the first is recognized by the depression and the bluish color; the second are pinkish or yellowish in color, while the emphysematous areas are pale in color and crepitate. The involvement concerns not only the small bronchi and the air-vesicles but extends into the peribronchial tissues and may there cause small septic foci. The exudate fills the vesicles and tubes, rendering them airless, and is composed of serum, red cells, epithelial cells from the bronchi and vesicles, leukocytes, bacteria and fibrin. This exudate finally undergoes resolution and its elements are absorbed or expectorated. Air again enters the occluded bronchi and air-vesicles as recovery takes place. Should this not occur connective tissue develops, which may finally result in a chronic interstitial pneumonia. Or an old or new tuberculous process may develop and the tubercle bacilli may later be found in the sputum.

Symptoms.—As broncho-pneumonia most frequently is a secondary disease, its symptoms resemble those of the primary affection. Whatever this may be there is usually a bronchitis of the larger tubes. Instead of this clearing up, the *fever* continues or suddenly develops and is accompanied by *marked increase* in the respirations (even to 50 or 60 per minute) and often by cyanosis. The *cough* becomes distressing, frequent and accompanied by expectoration. The pulse is accelerated and may become very feeble and irregular. The *primary form* may develop suddenly in adults and resemble acute catarrhal bronchitis. The *temperature* is moderate (101°-104° F.) and is irregular. The *cough* is attended with a tenacious *expectoration* and may be blood tinged.

Physical Signs.—Rapid and labored breathing and cyanosis may be observed. In children expiration is often prolonged and grunting. Cyanosis may be marked. *Percussion* shows dulness over the consolidated areas, though it may be masked by hyper-resonance. *Auscultation* reveals the characteristic subcrepitant and sibilant râles heard both during inspiration and expiration and distinguished best at the base and back of the lung.

Diagnosis.—The age of the patient, the gradual development of the symptoms, the preceding bronchitis, the absence of marked bronchial breathing, the bilateral involvement, the subcrepitant and sibilant râles, the increased respiratory efforts and the dyspnea are positive signs which should settle the diagnosis. The symptomatology of *acute simple bronchitis* is not confusing. *Lobar pneumonia* is usually unilateral, the chill and fever are more pronounced and the characteristic stages are well defined, while the pain of pneumonia is more marked and the previous signs of a bronchitis are not usually found. *Pulmonary edema* points more directly to cardiac or renal complications. *Acute phthisis* presents generally an apex consolidation, a more irregular temperature and the tuberculous cachexia may be present.

Prognosis.—The mortality ranges from 25 to 50 per cent. It is always a serious disease, especially in young children and in old people. Complicating diseases and faulty heredity make it more unfavorable. Persistent dyspnea, cyanosis and coma are grave signs, while cardiac disease or enfeeblement are serious complications.

Treatment.—The *general care* demands that the patient should be placed in bed and kept there. The temperature of the room should be maintained at 65° to 70° F. constantly, and the air may be moistened with steam in case of a persistent cough. A sufficiency of pure air is a constant and mandatory requirement. The cotton jacket may be applied to the chest and kept there until convalescence ensues, though the tendency of recent years has been to discard all chest applications. Hot fomentations are occasionally serviceable, but these, too, must be used with care not to excite the patient or encourage taking cold. A hot or possibly a tepid bath with gentle friction constantly maintained is essential in the beginning to encourage rapid toxic elimination. The bowels should also be thoroughly emptied and diuresis should be maintained by a constant and free use of pure water.

The *diet* should be light but nutritious. The rapid development of the disease may demand stimulants of some kind to tide over the depressing stage; but hot beef broth or whiskey added to milk may suffice. With children it may be best to repeat the hot bath, taking great pains after this to wrap them in a warm blanket. It seems almost unnecessary to urge caution regarding exposure to drafts, but this consideration is often overlooked.

The *remedies* in the early stages should be those suggested in simple bronchitis. *Tartar emetic* is most frequently indicated and covers a symptomatology indicative of the obstructive features; it also relates to the abundant secretion of mucus causing the rattling sound and the difficult expectoration; the third decimal potency, in four grain doses frequently repeated for a time, will give good results. In older patients, in which this potency does not cause improvement, it may be well to use a few doses of the second decimal potency, always being on guard lest nausea and vomiting are induced. The *arsenite of antimony* may many times favorably supplant this. *Belladonna* is very often indicated for the fever and congestive signs of the first stage, and it also answers well for the convulsive tendency which may be observed in children when the fever is high; the excessive thirst and the dry spasmodic cough, which is worse at night, should also call attention to this remedy. *Ipecac, kali bichromicum, antimonium crudum, sulphur, hepar sulphur, senega* and *opium* also have their sphere of usefulness. The employment of any opiate, however, should be considered only for the exhausting and obstinate cough as it is liable to retard the progress of resolution. *Bryonia* is often indicated in the early stages when pain is a factor and croupous symptoms are pronounced. *Asclepias tuberosa, sanguinaria* and *kali mur.* may be compared. *Silphium* aids the expectoration of tenacious mucus in much the same manner as *ammonium mur.,* which may be useful in the later stages. *Phosphorus, stannum* and *drosera* may be prescribed according to their indications. In this connection we may mention again the use of *apomorphia*. *Laurocerasus* has some efficacy in the spasmodic coughs characteristic of this disease wherein dyspnea and coughing spells, with much expectoration, are experienced. Under this remedy there is a constant feeling as if tenacious mucus were lodged in the throat and could not be loosened. *Creosotum* may be indicated when the symptoms of hoarseness and roughness are pronounced. *Sambucus* is a good remedy when the catarrhal invasion seems to affect especially the nasal and laryngeal mucosa. *Squilla, sanguinaria* and *spongia* may be required when the cough becomes extremely irritable and there is much dryness of the laryngeal and bronchial mucous membrane.

The necessity of using stimulants when cardiac weakness appears should always be remembered. For this purpose it may be well, at all times, to consider the use of *strychnia* (grs. 1/100-1/40). *Agaricin,* in the first decimal potency, will act well as a sustaining remedy for the weak heart in children. Caution should also be exercised toward keeping the patient extremely quiet, as in that way cardiac protection is observed and a more speedy reparation of the lung involvement is insured.

EMPHYSEMA.

This term properly applies to extravasation of air into the interstitial connective tissue of the lung. The *interlobular* and *vesicular* forms may be considered for the sake of study. The former is due to the escape of air into the interlobular connective tissue as the result of a rupture of air cells. Injuries, such as fractured ribs and penetrating chest wounds, violent exertion, violent paroxysmal coughing and hysterical convulsions may produce emphysema. The vesicular form is created by a dilatation or enlargement of the alveoli. There are three classes (1) compensating, (2) hypertrophic and (3) atrophic.

Compensating Emphysema.—This condition exists as a compensation for obstructions in other portions of the lung or in the opposite lung. Pulmonary tuberculosis, lobar pneumonia, adhesive pleurisy and cirrhosis of the lung lead to the development of a compensating emphysema in the healthy lung in order to maintain normal function. This condition is purely a physiological exaggeration and does not establish any pathological condition. The symptoms are not pronounced, except as they are observed in the abnormal functional activity of the healthy lung.

Hypertrophic Emphysema.—Substantive, idiopathic or the large-lunged emphysema of Jenner are the other terms given to this form of emphysema. It is characterized by the enlargement of the lungs because of the extreme distention of the air cells and the pathologic changes in the alveoli. It is also characterized by imperfect aeration of the blood and marked dyspnea.

Etiology.—The lung elasticity is lost on account of a persistent high intra-alveolar tension. The contractile power of the lung is at the same time depreciated so that permanent dilatation naturally follows. A congenital impairment of the contractibility of the alveoli, together with any unnatural pressure within the air cells may be given as usual primary causes. When the nutritive function of the air cells is impaired any increase of intra-alveolar air pressure may create the overdistention. This is particularly possible in violent attacks of coughing; the extreme muscular compression during a coughing paroxysm which causes an undue strain upon the weakened lung structures and the atrophic condition of the walls of the air cells permits an emphysematous distention. The sternum and costal cartilages gradually yield until the thorax becomes rotund. The disease may have its origin during childhood. The defect in elastic fibre development or congenital pulmonary weakness may be contributing factors. Asthma, bronchitis and whooping cough are frequently attending causes. Occupation should also be considered, as heavy lifting, violent muscular straining or the extreme lung activity demanded of players on wind instruments or glass blowers are liable to develop it. Heredity, no doubt, has its influence and advanced life must of necessity contribute a loss of lung elasticity which favors it.

Pathology.—The imperfect elasticity is the most characteristic sign; the lungs do not retract normally and are found at the autopsy encroaching upon the mediastinum. The color is changed to a pale gray with spots of dark pigmentation. The thorax is enlarged and the arch of the diaphragm is depressed. The substance of the lung tissue is soft and pits on pressure. On section it is found to be dry and bloodless. The dilated air vesicles vary in size. The septa are partially obliterated, the alveolar walls are thin and by perforation there may be a communication of air cells causing large air sacs. The elastic fibres become atrophied while the smooth muscular fibres are sometimes hypertrophied. The capillaries degenerate and the epithelium of the air cells undergoes a fatty metamorphosis. Beneath the pleura air bubbles may be found and in the anterior borders of the lungs air blebs or bullae are often discovered.

A bronchitis may be associated with emphysema and the bronchial structure is liable to manifest a similar degenerative condition. The heart is pushed down and back; hypertrophy and dilatation are observed in the right heart on account of the obstructed pulmonary circulation. The pulmonary artery and its branches become atheromatous. The liver and other abdominal organs suffer from a portal stasis, while the kidneys may show vital changes from the same cause.

Symptoms.—The characteristic symptoms appear in association with the predisposing disease, particularly bronchitis and asthma. *Dyspnea* is the most pronounced sign and this depends upon the degree of dilatation in the air cells. The respiratory effort is unusually emphasized. *Cyanosis* is a feature which appears as the result of cardiac and pulmonary insufficiency. *Cough* is another symptom of prominence and is noticed mostly in conjunction with bronchitic and asthmatic attacks. The patients invariably find that excessive exercise excites the dyspnea and other attendant symptoms. The dyspnea finally becomes continuous, with slight exacerbations. *Orthopnea* may exist and the labored respiration of asthma may frequently appear. In these conditions we find the expiration prolonged and labored; the accessory respiratory muscles are called into use and spasm of the bronchial tubes is liable to take place. *Circulatory obstruction* is evident by the turgidity of the face and neck. This disturbance of the systemic circulation may give rise to hemoptysis. Fatal hemorrhages have occurred.

The *general symptoms* of emphysema are manifest only after the condition has existed for some time. There is a gradual loss of flesh and strength, the patient is stoop-shouldered and there is evidence of a progressive cyanosis. Dilatation of the right ventricle is a frequent sequela.

Physical Signs.—The *barrel-shaped chest* is characteristic. The sternum and the costal cartilages bulge and the prominence of the infraclavicular and mammary regions give a peculiar rotund appearance to the thorax. The clavicles and cervical muscles are prominent and the stooping posture is due to the antero-posterior curvature of the spine. The intercostal spaces are widened and depressed. As the lungs are in a state of extreme expansion the respiratory action is vertical. The lower thoracic and upper abdominal regions may be retracted during inspiration. The respiratory effort is labored, the diaphragm and respiratory muscles undergoing violent activity. During the coughing spasm the supraclavicular lung area is excessively inflated, causing the so-called emphysematous tumor. There is also a marked increase in the antero-posterior diameter of the chest.

On *palpation* we find diminished vocal fremitus and a feeble or absent cardiac apex-beat. The engorgement of the right ventricle and cardiac displacement creates epigastric pulsation and a systolic shock over the ensiform cartilage. *Percussion* shows hyper-resonance which may at times be tympanitic. The cardiac and hepatic areas of dulness are greatly obliterated and the splenic dulness is somewhat lowered. By *auscultation* we discover a short and feeble inspiration and prolongation of the expiratory sound. The normal ratio of the two sounds is in a typical case exactly reversed. The expiratory sound is often harsh and may be accompanied by both coarse and moist râles, which are more distinct when bronchitis exists. In the interlobular variety a dry friction sound is heard. The pitch is somewhat lowered. The heart sounds are indistinct, though in case of right ventricle dilation a tricuspid regurgitant murmur is recognized.

Diagnosis.—The physical signs, the characteristic symptoms defined above, the long duration of the disease and the predisposing conditions of heredity and occupation will make the diagnosis certain. *Pneumothorax* is unilateral, motion is limited on the affected side, the percussion note is amphoric and the development is sudden. The slower course of emphysema, the absence of fever and the hyper-resonance should distinguish from *pleurisy with effusion,* which is usually unilateral.

Prognosis.—When emphysema is once established a positive cure is improbable. If taken in hand during the incipient stage some relief may be expected from measures directed to the inciting causes. Attending bronchitis, pneumonia, asthma or cardiac weakness will intensify the emphysematous symptoms. Renal complications make the prognosis more unfavorable. While the patient may live for years and the disturbing symptoms may from time to time improve, any intercurrent disease may terminate life.

Treatment.—Our first effort should be to care for and remove any contributing disease. Thus our attention will naturally be called to the treatment of attacks of *bronchitis* which invariably aggravate the emphysema. This will be accomplished by the measures and agents outlined under that disease. Any *occupation* which excites unusual lung activity must be given up. Any *hereditary tendency* must be considered carefully. The *diet* should be studied with a view to correct any digestive disturbance. Alcohol, irritating and rich foods must be interdicted. The *heart* should be protected against any unusual strain and if it shows any evidence of failing to do its work remedies should be immediately given to support it. The nervous system should be relieved from any unnatural excitement. In the chronic state the patient should be instructed in practical lung gymnastics and an outdoor life should be encouraged.

Asthma and violent paroxysms of coughing should be treated as previously outlined. *Phosphorus, grindelia robusta, ammonium mur., hepar sulph., ipecac, tartar emetic, silphium, drosera, calcarea iodid, kali hydriodicum, atropin* and *glonoin* will frequently be indicated. The heart may be sustained by *strychnia, spartein, digitalis, adonis* and *agaricin.*

Senile Emphysema.—This has been termed the small-lunged emphysema as it represents a senile atrophy of the lung parenchyma. The alveolar walls are atrophied to such an extent that the air cells coalesce and as a result large air sacs are produced. The normal amount of air in the lungs is reduced and the chest cavity is not filled by the lungs. The chest is contracted, the ribs approximate closely and none of the marked rotundity of the hypertrophic form is observed. The symptoms are decidedly negative and no treatment is of any avail.

GANGRENE OF THE LUNGS.

Etiology.—Pulmonary gangrene develops only when the lung tissue is sufficiently depreciated to permit infection by inhalation or through the pulmonic circulation. Therefore, malnutrition, renal diseases, diabetes or protracted fevers favor it. Lobar pneumonia, bronchiectasis or a tuberculous cavity may act as local causes, as may thoracic contusions, esophageal carcinoma, tumor or aneurysmal pressure or pulmonary embolism. An embolus from a gangrenous area is a common cause. Thrombosis of one of the larger branches of the pulmonary artery during typhoid may produce it. Foreign bodies are the most frequent causes and may enter the lungs during swallowing, by inhalation, and by perforation by malignant growths. The entrance of the bacteria of putrefaction is looked upon as a possible etiologic factor. Infective diseases of the mouth, pharynx or tongue and in fact any systemic poison, may be etiologic factors.

Pathology.—Pulmonary gangrene may occur in two forms —as a diffuse and a circumscribed process. The *diffuse* variety is rarely observed. It may appear secondarily to the circumscribed form or in conjunction with pneumonia or as the result of occlusion of a large branch of the pulmonary artery. When it exists an entire lobe or the greater portion of one lung may be involved in the gangrenous degeneration. The *circumscribed* form shows a well defined line of demarcation between the gangrenous and healthy tissue; there may be several similar foci in the same lung. The lower lobe is most frequently affected and the periphery is involved oftener than the central portion. The gangrenous area is greenish-brown in color, and as softening takes place a cavity containing greenish offensive fluid is formed. Outside of this there exists an area of extreme congestion or consolidation and still outside of this there is a zone of edema. Embolic gangrene is a frequent example and it is generally found in close proximity to the pulmonary pleura. Large vessels may be involved, thus causing hemorrhage and the pleura may be perforated, causing pyo-pneumothorax. Circumscribed abscess of the lung has a peculiarly intimate relation to cerebral abscess.

Symptoms.—The symptoms vary according to the primary process. *Irregular fever,* emaciation and profound prostration are observed but are not diagnostic. *Vomiting,* sweating, diarrhea and the pulmonary symptoms of the complicating disease may be evidence. *Severe pain* exists when the pleura is involved. *Dyspnea* is variable. *Hemoptysis* is liable to exist in certain cases. Cough may be present and accompanied by a horribly fetid and profuse expectoration. The *sputum* separates into three layers, the upper being frothy, mucopurulent and grayish-yellow in color, the middle layer is clear and watery, while the bottom layer is of a greenish-brown color, containing shreds of lung tissue, blood and pus.

The *physical signs* are not marked in obscure cases, but when the lesion is clearly defined the signs of consolidation are soon followed by the evidence of a cavity. *Bronchitis* is recognized and moist bronchial râles, which are often coarse, and cavernous râles are present.

Diagnosis.—The physical findings and the symptoms defined above, together with the horrible fetor of the breath and the characteristic sputum will be guiding indications. In *fetid bronchitis* the symptoms are more favorable and slower in development, while the fetor of the sputum and the breath is not so pronounced.

Prognosis.—A fatal result is liable to occur in the space of a week or ten days. There are rare recoveries from the circumscribed form. The causes of death are found in hemorrhage, exhaustion, peritonitis, pleurisy and pneumothorax.

Treatment.—The patient's strength must be sustained by stimulants and nourishing foods. Antiseptic inhalations have been suggested as valuable. *Creosote, iodin, carbolic acid* and *bromin* are useful for this purpose. The surgical treatment by incision and drainage has been reported as successful in a number of cases and our greatest hope lies in these measures.

ABSCESS OF THE LUNGS.

Etiology.—A circumscribed collection of pus in the lungs may exist in association with other diseases. There may be a predisposing condition when malnutrition is present. Streptococci, the diplococcus pneumoniae and Friedlander's bacillus have been found. A single abscess may develop from a fractured rib or other chest injuries. Pneumonia is a frequent cause. Perforation of the lung as the result of carcinoma of the esophagus, abscess of the liver or suppurating hydatids may result in pulmonary abscess. Multiple abscesses come from pyemic infection following emboli, hemorrhagic infarction, suppurating bronchial glands or other infections. Foreign bodies and purulent pleurisy are sometimes causes.

Pathology.—Pus and degenerated lung tissue are the characteristic pathological signs. The abscesses vary in size; a whole lobe of the lung may be involved and rupture into the pleura may take place, but more commonly they range in size from that of a filbert to that of a small orange. The walls are irregular and ragged but in old cases they may be composed of firm fibrous tissue. The suppurative process may occur as a diffuse purulent infiltration.

Symptoms.—The usual signs of pyemic infection may generally be recognized in the chills, the hectic fever and the persistent sweating. Especially valuable is the history of an antecedent pneumonia or septic process. The sudden discharge of a large amount of purulent sputum will aid in making the diagnosis. The *odor* is offensive, but not as bad as in gangrene of the lung or fetid bronchitis. Particles of lung tissue and elastic fibres may be found in the pus. The *physical diagnosis* may reveal the presence of consolidation and cavities if they are of fair size. This is possible only when the abscess is large. The *prognosis* is usually unfavorable, as the systemic infection is liable to be extreme before the abscess is discovered. Abscess following pneumonia is more favorable than any other form.

Treatment.—When abscess exists in the lungs we should resort to every means possible to sustain the patient's strength. Stimulation by whiskey is probably our best measure for this purpose. Tonics may be of some value in certain cases; sustaining food is a necessity. Antiseptic inhalations are recommended containing such drugs as creosote, thymol, etc., and may be serviceable. Surgery seems to be our best resort and this is particularly true when the abscess is situated near the surface. Such remedies as *echinacea, arnica, bryonia, hepar sulphur, sulphur, iodoform, guaiacol, silicea, cinchona* and *arsenicum iodid* should be considered, and may aid much in combating the symptoms as they develop.

PNEUMONOKONIOSIS.

This term is used to define those forms of lung fibrosis or chronic interstitial pneumonia due to the inhalation of dust in various occupations. *Anthracosis* is referred to as a coal miner's disease; *siderosis* is the term employed when the disease is excited by the inhalation of metallic dust and *chalicosis* is due to the inhalation of mineral dusts.

Anthracosis is a characteristic form of the disease and it is observed in its most typical form in those regions in which the atmosphere is well laden with the carbon particles. When the ciliary action of the mucous membrane is impaired these dust particles gain entrance to the lungs with the inspired air. These pierce the mucosa and reach the lymph-spaces and the lymph vessels and from this point they are conveyed to more remote points by the leukocytes. Arnold says that they are "first carried to the lymph-nodules surrounding the bronchi and blood vessels; to the interlobular septa beneath the pleura, where they lodge in and between the tissue-element; and along the larger lymph-channels to the substernal, bronchial and tracheal glands, in which the stroma-cells in the follicular cord dispose of them permanently and prevent them from entering the general circulation." In rare instances the carbon particles may enter the general circulation when the bronchial glands become adherent to the pulmonary veins. Chronic bronchitis succeeded by emphysema usually follows the primary anthracosis, though the presence of carbon particles in the protoplasmic cells may be the only changes observed in the lungs. A *chronic interstitial inflammation* follows as the result of the proliferation of the connective-tissue elements. This fibroid change starts in the peribronchial lymph-structures, and may involve the bronchial and tracheal glands. The affected part of the lung is coal-black in appearance, solidified and airless; the alveolar walls may be thickened and pleuritic adhesions may also exist. The areas of fibrosis vary, large portions of lung tissue often being implicated. Cavities containing a dark fluid may appear as the result of necrotic softening. Pulmonary tuberculosis is a common sequela of this disease.

Chalicosis is observed among those who work at stone cutting, or it may be found among the scissor and knife grinders. Interstitial changes similar to those of anthracosis take place in the pulmonary connective-tissue. Fibrous nodules having a gray center and dark periphery, and diffuse sclerotic areas are the characteristic phases of the pathology. These are dense and are cut with difficulty. The cut surface usually has a gray color, and a polished appearance.

Siderosis is recognized among those who follow the occupation of dyeing, ironsmithing, etc., the term referring to a collection of iron oxid in the lungs. Similar conditions may be noticed among grain shovelers, cotton spinners and cigar makers. The pathological changes are similar to those of the other forms of this disease, except the color of the affected areas is red instead of black.

Symptoms.—*Chronic bronchitis* is the characteristic condition which follows prolonged exposure to these exciting causes. In rare instances the onset may be characterized by an acute bronchitis. *Emphysema* soon succeeds the chronic bronchitis, while symptoms of asthma and dyspnea may appear. The *sputum* in anthracosis is dark, in chalicosis particles of silica will be found upon microscopical investigation, while in siderosis the expectoration is red in color. The mucopurulent characteristics of the sputum soon develop and the bacillus tuberculosis may be present later. The *physical signs* are those of bronchitis with emphysema to which may be added those of interstitial pneumonia and even cavity formation in some cases.

Diagnosis, Prognosis and Treatment.—The history of the case, the character of the sputum, the signs of bronchitis and emphysema and later the evidence of developing phthisis will usually suffice to make the diagnosis. The *prognosis* is only favorable providing climatic and hygienic changes may be made before the disease is well advanced. The pathologic condition favors the development of tuberculosis and new growths. *Treatment* should consider a change of occupation and a residence in a climate where pure air is obtainable. So far as remedies are concerned they are the same as are indicated in the different forms of bronchitis.

PULMONARY CARCINOMA.

Malignant growths in the lungs are generally secondary to a similar development in other parts of the body though they may rarely be a primary affection. All forms of carcinoma may exist. It may occur also by extension or contiguity.

Etiology.—The cause of primary cancer is the same as when it appears in other parts of the body. The female is afflicted more than the male, as secondary carcinomas most frequently follow a similar malignant growth in the breast. Extension of carcinoma, by continuity or contiguity or through the blood or lymph channels, is the source of development. The disease rarely appears before middle age. Heredity is claimed as a predisposing factor.

Pathology.—Encephaloid epithelioma and scirrhus are the usual varieties of the primary form. Of these, epitheliomas are more common. Primary cancer is generally unilateral and may involve the greater portion of one lung. The upper part of the lung and the pleura are most frequently involved, while the neighboring glands usually manifest a simultaneous invasion. The secondary forms are characterized by small and multiple nodules which are distributed throughout both lungs. Those of softer character display a degenerative condition in the central portion of the tumor.

Symptoms.—The symptoms present various features depending upon the degree of carcinomatous development and the location of the growth. *Pain* may be observed when the pleura is invaded. As the tumor develops respiration is interfered with and later *compression* may interfere with normal circulation. The compression may become so extreme as to invade the esophagus or press upon the recurrent laryngeal nerve, thus causing dysphagia or aphonia and hoarseness. Bronchitis is an attending symptom of great frequence and a constant cough may be in evidence. *Dyspnea* and *cyanosis* are present when the pulmonary function is depreciated or when compression obstructs the pulmonary circulation. Other symptoms resemble the general features of carcinomatous development.

Physical Signs.—These depend greatly upon the character, size and location of the tumor. The thoracic walls become unduly prominent in the case of large growths, and some evidence of a tumor may be noticed by inspection. Thoracic edema and enlargement of the cervical and axillary glands are valuable signs. The tactile fremitus is diminished or absent, the percussion note is flat and auscultation reveals friction sounds. The respiratory sounds are greatly weakened and bronchial breathing or signs of a lung cavity may exist.

Diagnosis.—The general history and the physical signs just mentioned will aid in making a diagnosis. *Pulmonary tuberculosis* is differentiated by the febrile and physical signs of that disease, but more particularly by the microscopic examination. *Fibroid induration* has a slower course.

Prognosis.—The inevitable tendency of this disease is toward asthenia, emaciation and death, the latter occurring in from six months to one or two years. It may be due to hemorrhage, asphyxia or asthenia.

Treatment.—As operative treatment is impossible our attention must be directed to the relief of symptoms as they appear. Possibly the X-rays may be of some value in giving relief from pain.

HYDATIDS OF THE LUNGS.

These occur generally as a secondary invasion from some other organ. The liver is most frequently the seat of origin and the lungs are invaded by perforation through the diaphragm or by way of the circulation. In addition to the symptoms which characterize the involvement of the liver we may observe pain in the chest, an irritating cough and sometimes bloodstained expectoration. The physical signs will show a lung consolidation or a pleural effusion. The vocal fremitus is diminished, expansion is imperfect, percussion shows dulness and the respiratory murmur is absent. To make a positive diagnosis we must find some evidence of the echinococcus in the sputum. The affection is incurable and may be attended by serious suffering as the growth attains large size. The treatment yields little hope unless an operation may be performed when the lung periphery is the seat of the lesion.

DISEASES OF THE PLEURAE.

PLEURISY.

Pleurisy or pleuritis is an inflammation of one or both pleural membranes and is subject to various classifications according to etiologic and pathologic conditions. It may, therefore, be considered as a primary or a secondary inflammation and may be characterized according to the infectious agent as tuberculous, septic, carcinomatous, etc. A pathologic differentiation recognizes a local or general pleurisy and again a plastic pleurisy or pleurisy with effusion. A further division according to duration gives rise to such terms as acute, subacute and chronic pleurisy.

The *cause* of pleurisy may be traced to some micro-organism, or its toxic product. The bacillus of tuberculosis, the streptococcus pyogenes, the staphylococcus, the diplococcus pneumoniae, the typhoid bacillus, the gonococcus, the ameba-coli, the bacillus of influenza and of anthrax as well as Friedlander's, and the colon bacillus have all been found in the exudate. In case of empyema we shall expect to find the micrococcus lanceolatus and the streptococcus as the particular causative agents.

ACUTE PLASTIC PLEURISY.

Etiology.—This is the dry or fibrinous form of pleurisy and may appear as a primary or secondary disease. In the primary form it is of rare occurrence and usually may be traced to a latent infection which is excited by some acute irritation. Exposure to cold, changes in the weather and traumatism are regarded as factors which induce it. The *secondary form* is more common and usually arises by extension from adjacent organs like the lungs. Therefore, pneumonia and broncho-pneumonia are usually the primary causes of this secondary condition. Other diseases of the lung like cancer, abscess, gangrene and hemorrhagic infarcts may induce it when the lung surface becomes involved. It is a frequent attendant of the chronic form of pulmonary tuberculosis, and may arise by extension from a hepatitis, pericarditis or even from a general peritonitis by way of the lymphatics. Chronic alcoholism and nephritis are also regarded as predisposing factors.

Pathology.—The affected part of the pleura shows a marked circumscribed inflammation, a dull and nonglistening surface, while minute ecchymoses are observed and as the exudate increases the surface becomes rough and shaggy. The exudate may become thick and have a yellowish-gray color. Numerous embryonic round cells also are included in its meshes, while blood vessels and connective tissue may constitute a part of the inflammatory product. Adhesion of the opposing surfaces usually takes place, though absorption of the exudate may occur in mild forms.

Symptoms.—In the milder forms the symptoms may not be marked, though the onset is usually announced by a chill or chilliness and *sharp, stitching pains* in the affected side. Inspiration and the slightest motion always aggravate the pain and for that reason the patient usually lies quietly upon the affected side or supports it with his hands. A dry and distressing *cough* usually attends, while the *inspiration* is short, hurried and painful. *Fever* is moderate (101°-103° F.) and not in proportion to the suffering. The *pulse* is small, soft and slightly increased in frequency. The more severe cases are ushered in by a pronounced chill, the pain is excruciating, the fever is high (104°-105° F.) the pulse is tense and erratic, while prostration is profound.

The *physical signs* are distinctive. *Inspection* will reveal the characteristic respiration and the restricted movement of the affected side, which may be confirmed by palpation. *Auscultation* will detect the friction sound which is most intense at the end of inspiration. When the fibrinous exudation takes place *palpation* will reveal decreased tactile fremitus while *percussion* shows a slight degree of dulness. *Auscultation* will disclose the peculiar friction or rubbing sound during inspiration and expiration, but most marked upon deep inspiration.

Diagnosis.—The friction sound plus the cardinal symptoms mentioned above are usually sufficient to establish a diagnosis. *Intercostal neuralgia* has many similar symptoms, but is not accompanied by fever and friction sounds, and is further characterized by the painful pressure-points. *Pleurodynia* may possibly be mistaken for the dry form of pleurisy, but the absence of a friction sound and other symptoms of pleurisy will differentiate it.

Prognosis.—This will vary in accordance with the severity of the attack. The ordinary case shows a subsidence of symptoms in a few days and the pathologic condition should disappear in a few weeks, under proper treatment. One attack naturally predisposes to another and repeated seizures may lead to pleural thickening and adhesions. The lung expansion is liable to be restricted for a long time unless proper physical exercises are employed. Repeated attacks of pleurisy always indicate an incipient tuberculosis.

Treatment.—*Pain* is generally so severe that morphin may be necessary for relief and for the same purpose it is essential to keep the patient in bed and in the position which assures the most comfort. *Local applications* are not always required, though nothing gives so much relief as the constant application of the icebag over the circumscribed spot. Some prefer hot applications. There is a wide difference of opinion as to the efficiency of antiphlogistic applications and poultices, though some still prefer them. Strapping the chest with adhesive plaster in complete expiration usually gives much relief. The internal remedies which are surely serviceable are *aconite, belladonna, veratrum viride, cantharis, byronia, capsicum* and *camphor*. We shall also influence greatly the inflammatory extension by the employment of *pulsatilla, calendula, hamamelis, arnica, hypericum* or other indicated remedies. Later *arsenicum iodid, iodin, iodoform, creosote, mercurius, hepar sulphur, asclepias, abrotanum* and similar remedies are required. When the acute symptoms have disappeared and convalescence is established, the patient should be compelled to practice deep breathing to regain as far as possible the normal function.

SERO-FIBRINOUS PLEURISY.

This is the most common form of pleurisy and is also termed according to its pathologic features, pleurisy with effusion or subacute pleurisy in which there is a variable amount of fluid exudate in addition to the fibrin.

Etiology.—Sero-fibrinous pleurisy may be a primary disease, though it is more frequently a secondary condition. The etiological factors are the same as in the dry form, but its severity depends largely upon the physical condition of the patient and the disease to which it is secondary. Exposure to cold and wet, injury, diseases of the lung and many of the acute infections are predisposing and possibly primary causes of the disease, but the direct cause can be traced to some micro-organism, the most frequent of which is the tubercle bacillus, it being present in about 75 per cent of these cases. This infection may originate in the pleura, but most frequently begins in the lungs. More rarely it develops secondary to a tuberculous peritonitis. While it is safe to assume that a majority of the cases of pleuritis are due to tuberculosis, the bacteriological examination of the sero-fibrinous exudate, according to Leube, rarely reveals the presence of the microorganisms. In isolated cases pneumococci, staphylococci, tubercle bacilli and streptococci are found in the characteristic exudate. Very frequently, however, the activity of the pyogenic bacteria causes the exudate to become purulent. In addition to tuberculosis, pneumonia and pericarditis as primary diseases, typhoid fever and rheumatism may predispose to this form of pleurisy.

Pathology.—The sero-fibrinous exudate is abundant, the greater part of the pleura on the affected side being involved. The fibrin is not alone found in the fluid, but is distributed also on the pleural surfaces. While the exudate is usually thin, it is sometimes very thick, forming creamy layers or curd-like masses on the dependent parts. It has a peculiar lemon color, though it may become dark brown. In the majority of cases it is clear or slightly turbid, depending upon the number of formed elements. When there are no adhesions the fluid always gravitates to the most dependent part of the pleural cavity. The amount of effusion varies from 1 to 8 pints. The microscope shows the presence of leukocytes, shreds of fibrin, red blood corpuscles and rarely cholesterin, uric acid and sugar. The fluid is rich in albumin, which may be detected by boiling, or may coagulate spontaneously upon standing.

Compression of the lung takes place in large and prolonged effusions and in extreme instances it may be crowded up and back against the vertebral column until complete atelectasis results. Other adjacent organs may also be compressed, the liver being crowded down in right-sided pleurisy; when the left side is affected the heart may assume more of a transverse position. Left-sided effusions are also liable to displace the stomach and transverse colon downward. Adhesions often prevent this displacement.

Symptoms.—In the majority of cases the *onset* is insidious, though in some cases it is sudden, as in the acute plastic variety. A *stitching pain* is first felt in the affected side, but this is not always so severe as in the plastic form. Many times it assumes a tearing or drawing character and is always excited by sudden movement, deep breathing and by sneezing and coughing. The intensity of the pain does not always indicate the severity of the condition; with the appearance of the effusion it gradually subsides. *Dyspnea* is one of the prominent features. The respirations are shallow, inspiration is "catching," so that cardiac disease is simulated. In case of copious and rapid effusion the dyspnea may increase even to orthopnea. *Cyanosis* is liable to be associated with the dyspnea, though there may be little respiratory disturbance when the effusion develops slowly. A *slight cough* is usually present and attending this there may be a little mucoid and blood streaked expectoration.

The *constitutional symptoms* are usually characterized by moderate fever (101°-103° F.) and a rapid, small and compressible pulse. In some instances the temperature is of the continued type. "There is a leukocytosis in non-tuberculous pleuritis, although the increase in the white cells is moderate." Anorexia, headache, weakness and constipation may cause the patient to consult his physician. The urine may be decreased in amount and of high specific gravity. There is a liability to aggravation or remission of the symptoms according to the character and extent of the effusion.

Physical Signs.—The early physical signs are identical with those of plastic pleurisy. *During the stage of effusion* it will be possible to elicit some distinctive signs of this form of pleurisy. *Inspection* discloses the restricted respiratory movement. As the effusion increases, bulging of the affected side is apparent with obliteration of the intercostal spaces in the middle and lower third of the chest wall; the apex beat is displaced in right-sided pleurisy to the left of the left mammary line, and in left-side pleurisy to the right of the right mammary line. In extreme right-sided effusions the apex beat may be pushed beyond the left vertical nipple line into the axillary space and as high as the fourth interspace. In left-sided exudations the heart's impulse is often not visible or it may be seen in the third or fourth right interspaces. The decubitus of the patient is also a characteristic sign, since this is usually assumed to relieve the pain. *Palpation* confirms the limited respiratory movement, the obliteration of the intercostal spaces and more accurately locates the apex. The most important sign determined by palpation is the diminished or abolished tactile fremitus, varying according to the amount of effusion. In children the fremitus may be obtained during crying. Adhesions which connect the pulmonary and the costal pleura may prevent diminution of the tactile fremitus. The displacement of the spleen and the liver are also determined by palpation. *Mensuration* is also a valuable adjuvant in physical diagnosis. The cyrtometer will detect a variance of one-half to one inch in the two sides in case of large effusions.

Percussion is the most valuable method for determining the presence and the amount of effusion. In the early stages the findings are not marked, but as the accumulation increases the resonance becomes defective until absolute flatness appears. The gradual increase in the height of the effusion will be determined by frequent percussion, the line of demarcation always changing with the position of the patient unless the effusion is circumscribed by adhesions. The upper limit of the fluid is generally higher posteriorly than anteriorly on account of the dorsal decubitus, though deviations may occur in every direction, the upper line of dulness often presenting a wavy outline. These variations are often due to adhesions between the pleural surfaces. In the subclavicular region a tympanitic note (Skoda's resonance) is commonly heard with pleural effusions. In copious exudations William's tracheal tone and the "cracked pot" sound may be elicited by percussion just below the clavicle.

Auscultation reveals the friction sound in the early stages, but this disappears as the effusion develops. The breath sounds are weak, distant or absent over the affected part, while bronchial breathing, increasing in intensity toward the upper border of the exudate and in some cases of a metallic character is characteristic. Bronchovesicular breathing is noticed above the level of the fluid, and the breath sounds of the opposite side are intensified. The auscultated voice is diminished in intensity over the effusion and bronchophony can be obtained when the compressed lung is adjacent to the thoracic wall. Egophony is frequently distinguished in pleurisy and is due "to the partial compression of small bronchi which causes the sound waves of the voice to pass the neighboring walls without obstruction." A friction sound may also be audible in places where the pleural surfaces are in contact.

The *stage of resorption* shows many changes in the physical findings. The affected side decreases in size, the intercostal spaces become normal and the respiratory movement gradually returns. Retraction may take the place of the previous bulging. This deformity is liable also to lead to a narrowing of the inferior intercostal spaces, a drooping of the shoulder on the affected side, while a spinal curvature, with the convexity directed to the sound side, may also be observed. *Palpation* will reveal the gradual return of tactile fremitus and the inspiratory movement of the chest wall. *Mensuration* will note the return to normal size in the affected side and *percussion* will detect the gradual yielding of the flat note to the normal percussion resonance. *Auscultation* will give the most confirming signs of improvement. The breath sounds gradually return as the effusion subsides. As the fluid disappears and the muscular tonicity is regained a friction sound will be detected as the two roughened pleural surfaces come into contact during respiration. These friction murmurs may remain for a long time and the compression may permanently affect the lower part of the lung, giving rise to a compensatory emphysema in the upper part. Another marked feature of auscultation in this stage is the gradual return of the normal heart sounds.

Other Clinical Varieties.—*Hemorrhagic pleurisy* is characterized by the visible presence of blood in the serofibrinous effusion. The causes for this are found (1) in the asthenic states accompanying malignant forms of infectious diseases; (2) in advanced age and alcoholism; (3) in tuberculous pleuritis following pulmonary tuberculosis; (4) in carcinoma of the pleura; (5) in Bright's disease and cirrhosis of the liver.

Diaphragmatic pleurisy is the term used when the inflammation is limited partly or entirely to the diaphragmatic pleura. The exudate may be plastic, serofibrinous or purulent, only a circumscribed spot being usually affected. Lancinating pain in the epigastric region is the most pronounced symptom. This may be increased by deep pressure over the point of insertion of the diaphragm at the tenth rib. Pain which extends from the anterior extremity of this rib to the sternum and xiphoid cartilage is considered as pathognomonic. Deep inspiration always augments the pain. The diaphragm is fixed, the respirations are short, and severe dyspnea, resembling angina, is often present. The dyspnea may increase even to orthopnea. A troublesome cough may be present, while nausea and vomiting are not uncommon. Fever is often a pronounced feature and the facial expression gives evidence of extreme suffering.

Encysted pleurisy is characterized by the circumscribed effusions due to adhesions. The effusion may be contained in separate pockets or loculi which often communicate. This condition may take place in any part of the chest and is exceedingly puzzling on account of the indefinite symptomatology. An exploratory puncture alone will settle the diagnosis.

Interlobular pleurisy takes place when the serous surfaces between the lobes are affected by the inflammatory process and the fluid becomes encapsulated on account of the interlobular pleural adhesions. On account of the small amount of fluid in the cysts there is only slight intercostal bulging and other suggestive symptoms are not in evidence. There may be a fistulous communication with a bronchus and the purulent expectoration may be the first sign of the true condition.

Diagnosis.—The symptoms which characterize acute plastic pleurisy are readily recognized, but in case of pleuritic effusion it is not always an easy matter to make a correct diagnosis. When there is a large effusion which displaces neighboring organs, causing bulging and immobilization of the affected side, it is not difficult to make the diagnosis. When, however, the effusion is not excessive and the physical signs are not pronounced, the diagnosis will be difficult. It is necessary always to decide whether the abnormality is an effusion, a retraction or compression of the lung. *Pneumonia* must be differentiated most frequently and, therefore, a thorough examination must be made. In *pneumonia* a short, sharp, and severe chill usually introduces the fever; in pleurisy the onset is marked commonly by a sense of chilliness which lasts for some time and rarely by a distinct chill. In pneumonia the pain is usually not so circumscribed or severe, while in pleurisy it is invariably sharp, "stitch-like" and localized. In pneumonia cough is an early symptom, showing the rusty sputum in the first stage; in pleurisy there is an irritating cough with little or no expectoration. In pneumonia the sputum reveals the pneumococcus while in pleurisy the sputum is either negative or occasionally reveals the tubercle bacillus. In pneumonia the fever is intense and declines by crisis; in pleurisy the fever is moderate and continuous, declining by lysis. In pneumonia the countenance is congested, herpes appear on the lips and face, leukocytosis is marked and prostration is pronounced. In pleurisy the face is pale, herpes are not present, leukocytosis is absent or slight and the prostration may not be so extreme.

The *physical signs* also display a wide differentiation. In pneumonia *inspection* reveals diminished mobility of the chest and no bulging, while in pleurisy with effusion thoracic distention is marked. On *palpation* in pneumonia the tactile fremitus is accentuated or only slightly diminished; it is never absent except when a bronchus is plugged and it is frequently intensified after a vigorous coughing spell. In pleurisy the tactile fremitus is diminished, suspended over an area of marked dulness, or entirely lost. On *percussion* in pneumonia the dulness is less complete and does not change in the altered position, the resistance to the pleximeter-finger is slight and occasionally a tympanitic note is observed; neighboring organs are rarely displaced in uncomplicated pneumonia. In pleurisy percussion flatness, with resistance to the finger, is marked, and the area of this flatness changes with the position of the patient, and if the effusion be large, neighboring organs are invariably displaced. *Auscultation* in pneumonia discloses râles in the first and third stages with bronchial breathing. In pleurisy the bronchial breathing is distant and rarely accompanied by râles; bronchophony may be detected, but egophony is frequent. The most characteristic auscultatory feature is the friction sound which is heard in the early and later stages. Aspiration in pneumonia yields nothing but a little blood while in pleurisy the serum is a positive evidence of the disease.

In *hemothorax* the history of trauma, aneurysm, pulmonary gangrene, etc., together with the sudden onset and the simultaneous symptoms of internal hemorrhage, usually rule out pleurisy. The diagnosis is, however, confirmed by obtaining blood by one or more exploratory punctures. *Hydrothorax* may closely simulate the effusion in pleurisy; this, however, usually furnishes a history of nephritis or cardiac disease, it is not associated with fever, the bulging is often bilateral and there is no pain or friction sound as in pleurisy. The specific gravity of the fluid in hydrothorax is less than 1015, while that of the pleuritic exudate is above 1017. *Tumors and cysts* of the thorax may present many simulating symptoms, but the history of the case, the location of the dulness on the middle or upper part of the lung and the irregular distention should settle the doubt. *Abscess of the liver* or an echinococcus cyst in this organ may also cause retraction or compression of the lung. The history and the character of the symptoms should naturally point to hepatic involvement. In either of these affections of the liver the upper line of dulness is usually fixed and exploratory puncture will yield a fluid which may be differentiated after careful examination. A *large pericardial effusion* is often confused with a left-sided pleuritic effusion. The history of a previous infectious disease is, however, generally obtained, dyspnea is a pronounced symptom and the heart is not displaced to the right. "Flat tympany is obtained in the posterior portion of the axilla and good pulmonary resonance at the base in the postero-lateral region of the chest, differing from the results of percussion in pleuritis." (Anders.) *Mediastinal* and *pleural tumors* are difficult to differentiate from exudative pleuritis. When, however, they grow from below upward the dulness extends slowly and in an irregular manner upward. Evidence of pressure on nerves, blood vessels or the esophagus is usually present, but aspiration is the most positive means of differentiation. *Peripleuritic abscesses* also closely simulate pleuritis with effusion, though the symptoms of lung compression and displacement of adjacent organs are usually absent, while the bulging of the intercostal spaces is generally restricted to the area of dulness.

In establishing the diagnosis of a *pleuritic exudate* it is essential to demonstrate whether it is simple or purulent. While the aspirating needle will decide whether it is a serous, purulent or hemorrhagic exudate it will be possible to recognize a pyothorax without this procedure. A septic temperature, intercurrent chills, sweating and general asthenia may usually be observed. We may also be suspicious of the purulent character of the exudate if it follows pneumonia, influenza or pyemia. While these features of diagnosis are always provisional, they are at least guiding features. The distinction between a strict tuberculous pleurisy and the rarer simple forms will call for a careful study of the symptoms and the clinical peculiarities of the case. "A high proportion of lymphocytes in the cells found in the effusion is indicative of tuberculous origin." (Anders.) Leukocytosis is also absent in tuberculous pleuritis. Osler says " a sterile fluid indicates in the majority of cases a tuberculous origin." A more positive diagnosis is made by the inoculation of a guinea pig with the exudate.

Prognosis.—This depends not alone upon the cause of the disease and the general condition of the patient, but upon the intensity of the attack and the involvement of adjacent organs. The proguosis is also influenced by the character and duration of the primary disease; if the pleurisy is a symptom of general tuberculosis, an unfavorable prognosis should be given. If it is only a simple serofibrinous pleurisy prompt evacuation of the fluid may permit an early resolution. If the liquid exudate is not withdrawn, or there is any mechanical hindrance to resorption, the disease may assume a subacute or chronic form or a fatal empyema may develop. A prolonged compression of lung tissue may create serious adhesions, or a chronic bronchitis or emphysema may supervene.

Treatment.—The early stages of this disease should receive the same therapeutic attention as mentioned under the treatment of the plastic form. *Limitation of the inflammatory process* is of first importance. For this purpose external applications of all kinds have been employed with the object of overcoming inflammatory extension by counter-irritation. Sinapisms, setons, blisters, cupping and poultices are not so universally used now as formerly. The icebag is probably the best local agent, both for the relief of pain and the limitation of inflammation. The mechanical fixation of the side by adhesive straps carried well around to the back from the center of the sternum, is a good expedient. Iodin persistently applied locally has probably a good influence during the early stages. It may be used in ointment or with ichthyol, or the tincture may be painted upon the skin. Iodoform cerate is worthy of trial when a tuberculous history is given. The *diet* in these cases should be of a light and nutritious character, limiting as much as possible the amount of liquids in order to favor absorption of the liquid exudate.

Internal medication has been employed with the hope of relieving or aborting the inflammation which causes the exudation. For this purpose *quinin, opium* and the *salicylates* have been extensively employed. In like manner diaphoretics, diuretics and salines have been used with the hope of securing a depletion which would limit or reduce the effusion. The homeopathic principle of administering remedies in accordance with the symptomatic expression of a pathology is certainly efficacious in this disease.

Aconite given according to its well known indications is one of our most valuable remedies in the early stages; *belladonna* frequently succeeds this and *arnica* may be demanded when traumatism is a factor in the development of the pleurisy. *Bryonia* and *cantharis* are most efficient remedies during the stage of effusion. It is a question which of the two remedies is the more valuable, but my preference is for cantharis, as I believe it will give more ready relief of pain and, to a certain extent limit the effusion. It is my experience, however, that it must be used in the tincture (m. 1-3) every two to four hours in order to obtain prompt relief. If the two remedies are compared it is my belief that bryonia will be indicated more particularly in the plastic variety of pleurisy, while cantharis is useful in the early stages of a rapid and profuse exudation. This will be characterized by a frequent cough, dyspnea, cardiac palpitation, profuse sweats, scanty urine and great prostration. *Apis mellifica* is often of great value when its characteristic symptoms are in evidence; not alone is the stinging pain an indication for its use, but it applies particularly to that class of cases in which edema is a distinctive feature and anemia is an underlying factor. Laryngeal edema is also a symptom of importance, and dyspnea, with a terrible sense of suffocation, is a common complaint. *Baptisia, lachesis* and *rhus tox* may be compared. *Arsenicum album* may be given when there is no response to bryonia, cantharis or apis mellifica and the patient is particularly exhausted and restless. *Arsenicum iodid* is a better form of the remedy in the slower developing effusions or when it is purulent or tuberculous. With this *kali iodid, calcarea phos., hepar sulphur* and *china* may be compared. When there is a rheumatic or gouty association we shall have recourse to remedies like *colchicin,* the *salicylates, rhus tox, ranunculus, stellaria media,* etc. When the pleurisy is plastic *kali carb., sanguinaria, stannum* and *squilla* may be demanded in place of bryonia. When the pleuritic effusion is secondary to pneumonia and there is that characteristic tightness which is incident to the early pneumonic stage *phosphorus* and *ferrum phos.* will be frequently indicated, while *spongia* and *iodin* are also useful. *Asclepias tuberosa,* or pleurisy root, which Hale defined as the "lesser bryonia," has a decided application in cases which are not extremely severe; it is not demanded when the fever is very high. In case of a purulent tendency there may be use for *sulphur,* the *iodids, silicea, calcarea fluorica, mercurius, tartar emetic* and similar remedies. It is, however, a serious mistake, in this stage, to rely entirely upon any remedy exclusive of surgical procedure.

If the exudate is not absorbed with comparative promptness it is best and essential to perform *paracentesis.* Every physician should be prepared to do this, always bearing in mind the necessity of strict asepsis. This operation is not only successful in the majority of cases but under the present aseptic methods it is free from danger. After proper preparation has been made with the patient in a semi-recumbent position, puncture with trochar or aspirating needle should be made in the mid-axillary line in the sixth interspace on the right side and the seventh interspace on the left side, or just below the outer angle of the scapula in the seventh right and eighth left interspace. The needle should be introduced with a sharp thrust close to the upper margin of the rib in order to avoid the intercostal artery, and the fluid should be withdrawn slowly. Thus the lung gradually expands and the heart does not suffer any sudden shock. If this precaution is not taken it is also possible for a sudden and fatal pulmonary edema to appear, following the congestion which arises from the sudden flushing of the paretic capillaries. The fluid is not always obtained at the first puncture inasmuch as the costal pleura may be excessively thickened, the fluid may be encapsulated or the needle may encounter a thickened fibrous band or be blocked by the exudate. If absorption does not take place after the first withdrawal of fluid, the operation must be repeated. When the effusion is directly associated with croupous pneumonia, particularly in the aged, it should not be evacuated. The operation should be stopped immediately in case of incessant cough, sudden edema, thoracic constriction, intense pain or syncope. After paracentesis it will be necessary to continue the indicated remedies and at the same time support the patient's strength by a judicious diet, rest, and the necessary hygienic measures. A tonic may be required the same as in the convalescing period of any disease. When the heart is in need of support we must supply that demand with our indicated cardiac remedies.

EMPYEMA.

Purulent pleurisy has been termed empyema or pyothorax, either of which refers to a suppurative inflammation of the pleura.

Etiology.—It may occur as a sequence of acute serofibrinous pleurisy, as the result of a purulent change in the fluid or possibly from the introduction of an unclean aspirating needle. The effusion is liable also to become purulent when children are affected, or it may be secondary to such acute and chronic infections as pyemia, scarlatina, pneumonia, tuberculosis, typhoid, measles, etc. Tuberculous cavities may perforate the pleura, lymphatic vessels may convey the infection from distant pyemic tissues, and traumatism following the fracture of a rib, or penetrating wounds are also possible factors. The *bacteriological examination* will reveal the presence of streptococci most frequently when the disease is secondary to a septic process, while pneumococci are usually found when pneumonia is the primary cause, and in such cases the disease generally pursues a favorable course. Staphylococci and tubercle bacilli are also present in some cases.

Pathology.—In post-mortem examinations the effusion is found separated into *two layers,* a clear, greenish-yellow serum above, and a thick or cream-like pus below. In a few cases the exudate is fibrinopurulent and in pneumonic empyema the pus is generally thick and creamy. The *odor* is sweetish, but in case of traumatism it is *fetid* and much more so when gangrene exists. *Microscopic examination* confirms the purulent character of the exudate and the pleural membranes are greatly thickened with granular suppurating surfaces. Perforations are common in old cases and the costal pleura may be the seat of erosions, while the lung tissue involved may be compressed.

Symptoms.—When the disease is of *acute character* the *onset* is sudden and is characterized by a severe rigor, high temperature, pain in the affected side which is always aggravated by deep inspiration and sudden movement. Extreme prostration usually intervenes very soon and dyspnea, with or without cough and expectoration, is an occasional symptom, due to pressure of the exudate. In the *chronic form,* which is more common, the symptoms are more obscure and insidious. The *general symptoms* are those of septic infection. Irregular chills, succeeded by fever, profuse perspiration, pallor and a gradual loss of flesh and strength, are usually distinctive. Should a gangrenous process ensue the symptoms may assume a typhoid character. *Peptonuria* is a common symptom and *indican* is frequently found in the urine. A *leukocytosis* will be revealed by blood examination. *Perforation* may take place into the lung, giving rise to pneumothorax or the rupture may occur into the pericardium, peritoneum, esophagus or stomach.

The *physical signs* are, to a great extent, the same as those in pleurisy with effusion. There is often edema of the chest wall, the subcutaneous veins are quite distinct, the intercostal spaces of the affected part are liable to bulge, local redness and fluctuation are occasionally noticed and the abscess may, if not aspirated, come to a focus and rupture externally. These features are always most pronounced when children are afflicted. In children also the breath-sounds are inclined "to be loud and tubular over a purulent effusion of considerable size." The upper area of dulness changes less promptly than in the case of serous effusion when the position is altered. *Baccelli's sign,* the transmission of the whispered voice through a serous exudate, may aid in differentiating serofibrinous pleurisy from empyema. Senator also suggested that there is a *greater displacement* of the heart and liver in empyema on account of the greater weight of the fluid. *Pneumothorax* is also liable to attend empyema, particularly in case of lung perforation.

Diagnosis.—The physical signs and many of the symptoms are so similar to those of pleurisy with effusion that an accurate diagnosis is only made by means of paracentesis to determine the character of the exudate. When pulsation is present the effusion may be mistaken for an *aneurysm of the thoracic aorta.* The absence of the vascular symptoms of aneurysm, the location of the pulsation some distance from the normal aorta, together with the evident physical signs and symptoms of empyema will make the differentiation.

Prognosis.—The disease is always a serious one. Incomplete absorption of the purulent fluid is usual unless surgical measures are employed and it may be a source of grave general infection. Rupture, externally or into a bronchus, is equally unfortunate. A favorable diagnosis, therefore, depends entirely upon the surgical evacuation of the purulent fluid and in the majority of cases which are operated upon with aseptic care a cure is accomplished. If this measure is not adopted we may expect our patient to die from gradual asthenia. Associate conditions like phthisis and pericarditis invariably make the diagnosis more unfavorable. Double empyema is always serious. Children respond to surgical treatment with marked success. Obliteration of the pleural cavity by adhesions is liable to take place and in such instances there may be retraction of the affected side. This, however, may be overcome by the employment of proper exercises after the operation. It has been said that the spontaneous evacuation through the chest wall favors a cure, but there is no excuse for any physician to allow a case to reach this stage.

Treatment.—The treatment of empyema is purely surgical. The patient having been prepared as for any operation, the chest wall is opened under local or general anesthesia in the fifth or sixth interspace in the anterior, and in the eighth or ninth interspace in the posterior axillary line. If free drainage can not be obtained by simple incision, resection of one or two ribs is demanded. The cavity should not be irrigated unless the fluid has a foul odor. Normal salt solution or a 2 per cent boric acid solution are preferably used. Drainage tubes should be inserted, using those which will not collapse. Abundant dressings should cover the wound and these should be changed as soon as they become saturated with the discharge. Special measures should be adopted to obliterate the cavity, to prevent adhesions and to develop the collapsed lung.

CHRONIC PLEURISY.

Chronic pleurisy occurs in two forms with effusion and without effusion. *Chronic pleurisy with effusion* may be of insidious development or it may follow an acute serofibrinous attack. In such cases the fluid remains a long time in the pleural sac without undergoing any special change. When it is large in amount the surrounding organs may be pushed out of place. When the fluid is absorbed or removed, contraction of the affected side takes place on account of the organized adhesive bands which prevent expansion of the contracted lung. The *symptoms* are not pronounced and aside from the slight dyspnea and cardiac excitement upon exertion there are few subjective symptoms. Should the effusion become purulent we may expect hectic temperature, sweating and other signs of septic fever, terminating in general asthenia. Such a development is always dangerous and abscess of the brain may also be a secondary and fatal termination of the disease. The physical signs are similar to those of serofibrinous pleurisy. The disease may remain in this chronic stage for a long time or the patient may die from some intercurrent disorder.

The *chronic dry or adhesive pleurisy* may follow the acute or chronic serofibrinous form, in which case the liquid exudate is absorbed. The fibrinous portion of the exudate becomes organized until the thickened pleural layers approximate and form firm adhesions. There is another form of chronic dry pleurisy which is purely idiopathic inasmuch as there is no history of acute symptoms. The patient, in these cases, is only warned of the pleurisy by the sense of constriction. Adhesion of the pleural layers also exists, though the exudate is usually so slight that the fibrinous attachment is circumscribed. *Retraction of the lung* in chronic dry pleurisy is not usually so pronounced when the pleuritis is a sequence of the acute form; in this instance the fibrous bands are not so firm or extensive that they decidedly interfere with proper lung expansion. If, however, the chronic form of serofibrinous pleurisy precedes, or if empyema is the original cause, the retraction will be extreme. The exudate may also undergo calcification. In the idiopathic form the circumscribed adhesions may be of tuberculous origin and in such instances tubercles may be found in the thickened pleura. General synechia may be present on one or both sides.

Symptoms.—Characteristic symptoms are rarely present and except for the restricted expansion of the affected side and the feeble respiratory murmur, the disease might pass unrecognized. The *physical signs* are by no means reliable or conspicuous. Some cases will clearly reveal the immobility and contraction of one side compared to the exaggerated action and distension of the opposite side, while the heart may be conspicuously displaced. In some cases inspection may disclose a spinal curvature, a drooping shoulder and a retracted thorax. The tactile fremitus also may be feeble or lost over the affected area, together with impairment of percussion-resonance. In rare instances the weakness of the breath sounds are out of proportion to the chest restriction, while occasionally a friction sound is audible. Vasomotor disturbances are occasionally observed and serious cardiac disorders are sometimes present.

Treatment.—Should there be any effusion surgical measures are required for its evacuation. Aside from this it will be necessary to employ remedies in accordance with the symptom-complex, and to sustain the nutrition of the patient. *Lung gymnastics* should be carefully instituted and persistently practiced. A climatic change is often required and the physician should always be on guard lest pulmonary tuberculosis develop. If tuberculosis is the underlying disease the patient should be treated with special reference to that condition.

PNEUMOTHORAX.

Pneumothorax is that uncommon condition in which air is found in the pleural cavity. The term sero-pneumothorax is also used when serum is present; when the fluid assumes a purulent character the term pyo-pneumothorax is employed.

Etiology.—Among the *predisposing causes* we may mention age, as older people are more frequently afflicted than children, and sex, inasmuch as males are more predisposed to injury. The left side is the most frequent seat of the disease and emphysema in which the "superficial air-sacs are dilated and atrophied" may also predispose to it. Of the *exciting causes* the most prominent factor is a perforating wound which lacerates the lung and pulmonary pleura. Fracture of the ribs may produce pneumothorax, while the rupture of a tuberculous cavity into the pleural cavity is a more common cause. Rupture of the air vesicles during violent muscular effort has occurred in some cases. When there is a necrotic condition of the lung due to such conditions as gangrene, bronchopneumonia, suppurating bronchial glands, pulmonary abscess and echinococcus cysts pneumothorax may develop. Perforation of the diaphragmatic pleura by malignant diseases or ulcer of the stomach or colon, or as the result of abscess or malignant diseases of the esophagus, are occasionally etiological factors. A peripheral bronchiectasis may likewise rupture into the pleural sac and a perforating empyema may involve the visceral pleura, the lungs and bronchi. A "gas-forming bacterium" sometimes produces pneumothorax by its action on the pleural exudate.

Pathology.—The point of perforation is often discovered to be the rupture of a tuberculous cavity or callous tubercle. Sometimes it is impossible to locate the seat of rupture or to determine the cause. Occasionally it is possible to trace a fistulous connection between the pleural sac and the bronchus. The lung is compressed and carnified, the heart is pushed to the opposite side, thought the relation of its parts is unchanged. The spleen is also liable to displacement in left-sided pneumothorax. A circumscribed pneumothorax may exist when firm adhesions limit the introduction of air. A purulent or seropurulent effusion is usually found in the pleural cavity, and is generally accompanied by inflammatory changes.

Symptoms.—The *onset* is usually sudden and is characterized by agonizing pain in the affected side. *Dyspnea* with intense suffocation and *cyanosis* follow promptly, the severity of these symptoms depending upon the amount of air entering into the pleural sac and the degree of lung compression resulting. The *respirations* are rapid, the *pulse* is frequent and feeble, the *temperature* may drop at first, owing to the collapse, but soon rises and assumes a hectic type due to consequent pleuritis, which is often purulent. Edema of the hand on the affected side has been noticed. The character of the symptoms depends largely upon the *rapidity* with which the air enters the pleural sac and the previous condition of the pleura and lung. In some instances the symptoms are much modified, particularly in cases of chronic phthisis.

Physical Signs.—*Inspection* shows marked enlargement and immobility of the affected side and unnatural mobility of the unaffected side. *Palpation* reveals diminished or absent tactile fremitus, and a feeble and displaced cardiac impulse. The *percussion note* is resonant, often ringing and amphoric over the part containing air, while over the area containing fluid there is marked dulness. On account of the extreme tension which checks all vibration there may be dulness over the air-containing space. Biemer's change of note or modification of tympany in proportion to the size of the air-containing cavity under a given tension may be observed. A "cracked pot" sound is also present when the pleural cavity freely communicates with the external air. Wintrich's sign, or the change in pitch of the percussion note when the mouth is opened, may also be detected. If the patient assumes the upright position, the air-containing space is enlarged as the weight of the fluid pushes the diaphragm downward. On account of the changed position of the heart there is usually resonance over the normal cardiac region, particularly when the patient is in the recumbent position. The liver and spleen are displaced according to the side affected and, hence, the normal percussion note over these organs is changed. *Auscultation* recognizes the feeble or absent vesicular murmur over the part affected and amphoric breathing is audible in case of open pneumothorax. "Metallic tinkling," a sound ascribed to the dropping of fluid from the seat of perforation into the fluid below, is occasionally heard. "Coin clinking" is also a pathognomonic sign; this is heard with the ear of the auscultator at the back of the chest, while a coin placed over the affected side in front is tapped by another coin. The listening ear will detect the intensified echo of the coin sound. The "Hippocratic succussion" is another characteristic sign which is elicited by shaking the patient while the ear is applied to the chest, a distinct splashing sound being heard.

Diagnosis.—The diagnosis must be made with a full knowledge of the history and the presence of the distinctive physical signs. The "coin-sound" and the "succussion-splash" are the most positive physical signs while those elicited by inspection, palpation and percussion must be taken into careful consideration. *Large tuberculous cavities* in the lung may closely simulate the disease; in these cases there is immobility, but the chest is flattened instead of bulging and the apex beat is not displaced. Fremitus is increased or normal, while in pneumothorax it is diminished or absent. Percussion gives tympany or a "cracked-pot" sound, Wintrich's sign being more pronounced in cavity, but the coin test and the succussion sounds are not present as in pneumothorax. Bronchial breathing, increased vocal resonance and the presence of râles are also detected in pulmonary cavity. *Bronchiectatic cavities* may be differentiated by percussion dulness instead of tympany, the absence of bulging, the distinct vocal fremitus and pectoriloquy which is never present in pneumothorax. *Diaphragmatic hernia* closely resembles pneumothorax in its physical signs and symptoms. Hernia, however, may appear and disappear suddenly. *Distension of the stomach* may cause some confusion, but the limitation of the physical signs to the region of the stomach and the absence of thoracic distention will overcome this mistake. *Subphrenic* abscess is usually on the right side, involving the diaphragm above the liver. It is usually attended by ulceration of the stomach and circumscribed peritonitis. The heart is not displaced and the bulging is entirely in the hypochondrium.

Prognosis.—The disease is usually incurable and often suddenly fatal. Much depends upon the cause. In advanced phthisis it is exceedingly unfavorable. Those cases occurring from trauma, lung abscess or emphysema sometimes recover.

Treatment.—The *relief of pain,* aside from the use of cardiac stimulants, is the first object of treatment. This is accomplished best by the hypodermatic use of morphin or codein. *Operative procedure* is the next requirement and if there is a serofibrinous effusion, aspiration is demanded but if the effusion is purulent, perfect drainage must be obtained by costal resection the same as in empyema. When the pneumothorax exists in connection with phthisis such radical measures are rarely advisable. The air chamber above the fluid is sometimes tapped by an aspirating needle, and Unverricht, in cases of pulmonary fistula, has inserted a tube into the pleural sac, thus allowing free entrance of air, the complete collapse of the lung and closing of the fistula.

HYDROTHORAX.

This is an accumulation of transuded serum in the pleural cavity. The transudate is a clear amber-colored liquid free from fibrin, though cholesterin and a few endothelial cells may be found in it. It contains less albumin than the pleuritic exudate. In reaction it is alkaline, the specific gravity is low (1008-1012) and the fluid is considered to be non-inflammatory in character. The pleural surfaces are smooth, though somewhat pale and edematous. The hydrothorax is caused by a resistance to the free circulation of blood throughout the pleura, and it is most frequently the result of chronic nephritis and valvular heart disease. It is also secondary to chronic dysentery, diarrhea, leukemia, pernicious anemia, carcinoma, malaria, syphilis and scurvy. Carcinoma of the pleura, compression of the superior vena cava or thoracic duct from any cause may develop it.

Symptoms.—The *general symptomatology* partakes of the primary disease and the symptoms directly due to the hydrothorax are those which are incident to the mechanical pressure. Thus *dyspnea,* often terminating in *orthopnea,* and *cyanosis* are the most pronounced signs, while the evidence of a *feeble circulation* and a troublesome *cough* are quite constant. In many instances the patient is subject to respiratory disturbances which resemble attacks of asthma. The *physical signs* resemble those of pleuritic effusion, though crepitant râles due to retraction and partial atelectasis may sometimes be heard above the effusion. *Hemothorax* is a condition in which blood takes the place of the serous effusion following an aneurysmal rupture, wounds of the bloodvessels and malignant disease. The physical signs and symptoms are similar to those of hydrothorax.

Prognosis and Treatment.—The condition of the patient, the character of the primary disease and the promptness of the surgical relief affect the prognosis. So far as the transudate itself is concerned its safe removal is possible. The general treatment is in line with that of pleurisy with effusion though strict attention must be given to the cause which induces it.

NEOPLASMS OF THE PLEURA.

Morbid growths which affect the pleura are exceedingly rare. Both carcinoma and sarcoma may exist, their clinical phenomena being identical. *Carcinoma* is a secondary disease which affects the pleura by direct extension of primary cancer of the lung. Secondary cancer may occasionally arise by metastasis from the mammary gland or from the lungs. Primary carcinomatous involvement of the pleura is an exceedingly rare condition.

The *pathologic findings* reveal circumscribed areas of thickening or the neoplasms may assume the form of papular projections often developing into pedunculated enlargements. In size the growths may vary from that of a pea to that of an apple. The neighboring pleura is thickened, often adherent and effusion may develop. The *fluid* attained by puncture is usually hemorrhagic in character with a color varying from red to reddish-black. It contains an abundance of fatty degenerated cells and large endothelium with vacuoles due to the degenerative process of the cancer cells. *Sarcoma* occurs sometimes as a primary growth "in the shape of the so-called endothelial carcinoma of Wagner, which starts from the endothelial cells of the lymphatics and connective tissue." Secondary sarcomatous deposits may be found in the lungs, lymphatic glands, the liver and muscles.

The *symptoms* of any one of these forms are not pathognomonic, as they may resemble those of mediastinal tumors and chronic pleuritis. The collective symptomatology supported by the rapidly advancing cachexia and the demonstration of cancer in other parts of the body are the only objective signs which suggest a diagnosis. Often it is impossible to recognize the disease until the extreme loss of flesh and strength and the discovery of nodules in neighboring localities point to the true condition. The hemorrhagic character of the effusion will help to differentiate from the ordinary pleuritic effusion. *Pain* is always a more constant feature than in the simple chronic pleurisy. *Chondroma, lipoma* and *calcification* of the inflammatory pleural products are also occasionally observed. *Echinococcus* or *hydatid disease* is a rare pleural involvement. The presence of hydrothorax, with its non-albuminous fluid, and the discovery of hooklets and fragments of the hydatid cyst in the aspirated fluid are distinct differentiations, while the purulent character of the fluid is also diagnostic.

The *prognosis* is always unfavorable and the treatment is only palliative.

DISEASES OF THE MEDIASTINUM.

With the exception of diseases of the heart, aorta, trachea and esophagus, mediastinal disease includes all those morbid conditions which pertain to the mediastinal space. Tumors, inflammation, diseases of the thymus gland and mediastinal hemorrhage may be considered.

Tumors of the Mediastinum.—*Sarcoma* and *carcinoma* constitute the majority of these neoplasms, the former being most frequently observed. Lymphadenoid tumors, fibroma, dermoid cyst and hydatid cyst are of occasional occurrence, while enchondroma, lipoma and gumma are even more rare. Primary sarcoma is more frequent than primary carcinoma and it may start from the "remnant of the thymus gland, from the lymphatic glands, the pleura, the lungs or from the fibrous tissue of the mediastinum." The esophagus, bronchi, lungs and rarely the thymus gland may be the primary seat of its first development. Carcinoma is most frequently secondary to carcinomatous invasion of the lymphatic glands. Mediastinal carcinoma is rarely primary. Females are most commonly affected and it occurs usually in advanced life.

Symptoms.—Aside from the vague symptoms of cachexia, slight dyspnea and some substernal pain, the early symptoms are not indicative of the developing neoplasm. When pressure becomes a factor, *pain* will be marked and constant. This is not always severe, though it persists and gradually increases in severity. It usually radiates to the sides of the chest and many extend down the arm. *Dyspnea,* slight in character at first, soon becomes a distressing feature. In the early stages this may assume an asthmatic character. It is incident to pressure upon the trachea, bronchus or the recurrent laryngeal nerve. *Cough* and aphonia are usually present. *Dysphagia* will exist when the esophagus is the seat of most intense pressure. Pressure upon the vagus or the sympathetic will give rise to cardiac disturbances. *Compression* of the superior vena cava or subclavian will induce cyanosis and edema of parts in which the venous circulation is suppressed. If collateral circulation is not well established, dilatation of the superficial veins may result from the venous obstruction.

The *physical signs* are, on *inspection,* a diffuse and irregular swelling in the sternal region. If extensive erosion takes place, the tumor may be recognized immediately beneath the skin. It is only in the advanced stages that any sternal prominence is noticed. *Palpation* detects the feeble or absent fremitus over the growth and a pulsation may indicate the heart's apical impulse. *Percussion* elicits dulness even in the earliest development, but this will vary in accordance with the size and growth of the tumor. *Auscultation* is rather negative though the breath sounds and vocal fremitus are feeble or absent while, in rare instances, a bruit is heard over the seat of the tumor. Should pleural effusion attend the morbid development additional physical signs will be recognized.

Diagnosis.—A careful study of the history and outlined symptoms will be of guiding significance though it is always necessary to exclude the tumors and diseases of neighboring organs and tissues. *Aneurysm,* with its softer tumor, longer course of development and the strong, heaving and expansile pulsation, will usually be differentiated from the solid mediastinal tumor. The severe diastolic shock which is detected in aneurysm is absent. The X-ray may help in substantiating the evidence of a morbid growth.

The *prognosis* is always unfavorable and *treatment* avails us nothing. The duration of the disease is from six months to a year and one-half.

Inflammation Affecting the Mediastinum.—Inflammatory involvement of the mediastinal glands and connective tissue is of occasional occurrence. *Lymphadenitis* of moderate grade is occasionally observed with broncho-pneumonia and bronchitis following such infections as measles, influenza and whooping cough. Tuberculous lymphadenitis has been mentioned under its appropriate head. Suppuration of the mediastinal lymph glands may take place as the result of a local specific infection. This is not generally recognized unless there is perforation of the esophagus or a bronchus and then, as in all cases of this kind, a fatal result is to be expected. Fortunately, *abscess of the mediastinum* is of rare occurrence and it is most frequently located in the anterior part. Traumatism and such infections as erysipelas, rheumatic fever, measles and smallpox are the common causes. Suppurative extension from neighboring parts and pulmonary tuberculosis are marked factors in this form of mediastinal involvement.

The *symptoms* of acute abscess will be characterized by *pain* and tenderness in the sternal region, while *cough* and *dyspnea* are usually present. The physical signs consist in percussion dulness which increases with the abscess development, while palpation may detect pulsation and fluctuation. The abscess may, by the process of burrowing, invade the trachea, esophagus or the abdomen. In *chronic abscess* the symptoms may simulate more closely those of a solid tumor. In case of a spontaneous cure absorption and inspissation take place.

Diagnosis.—In *acute abscess* the sudden onset, the rapid course, and the suppurative character of the symptoms will establish the differentiation from aneurysm, while palpation will distinguish the more solid tumor. An exploratory puncture will confirm the diagnosis. The *treatment* is purely surgical.

Mediastinal Hemorrhage.—The connective tissue of the mediastinum in rare cases is liable to hemorrhage from rupture of aneurysms of the arch or of the large vessels within the thorax. Traumatism is also a possible factor in this respect. The hemorrhagic diathesis is also a predisposing cause, though it is difficult to differentiate between this and hemorrhage into the thymus gland.

Diseases of the Thymus Gland.—The thymus gland may be subject to hypertrophy, organic growth or abscess, though it is extremely difficult to detect the difference between these and mediastinal involvement of the same character. Sudden and fatal dyspnea, as the result of pressure, is about the only substantial symptom. Laryngismus stridulus and the so-called "asthma thymicum" are suspicious symptoms when this gland is enlarged.

DISEASES OF THE HEART AND BLOOD VESSELS.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

This is a disease which is characterized by an inflammatory invasion of the pericardial sac. To understand it properly one must be familiar with the anatomical structure of the pericardium. This, we must remember, is a cone shaped sac, the base of which rests upon the diaphragm while its apex extends upward, surrounding the cardiac vessels. The external layer is thick and fibrous, while the inner layer is thin and serous in character. Areolar connective tissue binds the pericardium to the heart muscle. The pericardial sac, which is almost entirely covered by the pleura, contains a small quantity of alkaline fluid. The varieties of pericarditis are described as acute, plastic or fibrinous, serofibrinous, purulent, hemorrhagic and adhesive. A tuberculous pericarditis is also recognized.

General Etiology.—*Primary* or idiopathic pericarditis rarely, if ever, exists. It is supposed to occur in children when there is no positive sign of infectious origin, but even this form may be subsequently traced to a bacteriological cause in which the bacillus tuberculosis is a frequent factor. Traumatism may possibly have an influence in inducing the disease, but an infectious agent will usually be found. Inflammatory extension by contiguity from adjacent organs may sometimes be observed. Pleurisy, pneumonia and inflammatory invasions of the esophagus or bronchial glands are most prominent in this respect.

As a *secondary* process we shall find that rheumatism is a most prolific cause. In fact, pericarditis may complicate almost any stage of rheumatic fever, and may occlude or overshadow the typical symptoms. We also find it associated with acute tonsillitis and chorea. It may also develop as a complication or as a sequela of scarlet fever, influenza, pneumonia, pyemia, variola and other infectious diseases, while scurvy and diabetes sometimes predispose. It may occur in the later stages of parenchymatous nephritis, particularly in old people; as in this case it attends a disease of insidious character, the physical signs are not always detected with ease. Pericarditis, secondary to general tuberculosis, will, doubtless, be more fully recognized as pathologic investigations become more perfect. That it results from the tuberculous invasion of the mediastinal glands there is little doubt; and pulmonary tuberculosis in all probability is a common contributing source of infection.

ACUTE PLASTIC OR FIBRINOUS PERICARDITIS.

This form of pericarditis is the most common variety and rarely proves fatal unless unforeseen complications attend. It is almost invariably a secondary condition and is characterized by the small amount of fluid exudate and the predominance of the fibrinous exudate.

Pathology.—The *membrane* at first is smooth, swollen and injected; ecchymotic spots may appear in certain areas. The pathologic process may show local or general distribution. The *exudate* may simply appear as a roughened condition of the pericardium, or it may, in the severer types, create a pronounced thickness of that membrane. As this exudate becomes organized the constant friction of the opposing surfaces develops a peculiar shaggy appearance which has been defined as the "hairy heart." In other cases the exudate may appear in the form of ridges, or the pericardium may seem to be honeycombed. In *prolonged cases* the myocardium is liable to be affected, giving a pale and turbid appearance to the muscle. Tubercular granulations are often found in this form of pericarditis and a slight serous effusion will be detected at times.

Symptoms.—The symptoms are not always prominent and are mostly indicative of the complicating disease. The pericarditis may be detected only at the autopsy. When symptoms are observed the patient will complain of a *sense of distress, constriction,* or *slight pain* in the precordial region. The pain is most marked prior to any effusion and extends down the left arm, into the back, or it is reflected to the ensiform cartilage or the abdomen. *Pressure* over the pericardium does not increase the pain in the average case. *Anginal* symptoms sometimes develop. *Palpitation* and *dyspnea* are generally detected and the *pulse* is increased in frequency; it becomes weak in the later period of the disease or when the myocardium is affected. Slight fever may exist, though this depends much upon the complicating disease.

Physical Signs.—*Inspection* reveals increased vigor of the apex beat. *Friction fremitus* is felt at the base during the early period of the disease. *Percussion* is negative. *Auscultation* gives a distinct friction sound which is superficial and is intensified by the pressure of the stethoscope. It has a to and fro character and loses its distinctness by deep respiration and postural change. The quality of the sound varies; sometimes it is soft, but more commonly it has a grating, rubbing or creaking character. With few exceptions these adventitious sounds are double; they attend the rhythmic movements of the heart, but are not generally synchronous with the heart sounds as their duration is longer. The seat of maximum intensity is in the fourth and fifth interspaces to the left of the sternum. The sound may be most marked at the base, or over the ascending aorta, and sometimes it is easily detected at the apex. It may be heard over a small area, or it may be transmitted up and down the sternum.

Diagnosis.—A careful study of the physical signs enumerated above will aid the examiner in making a proper diagnosis. We must differentiate between this and the *pericarditis with effusion* by the characteristic signs which show the presence of a fluid exudate. *Aortic incompetency* may simulate the pericardial rub, but it will be distinguished by the constant character of the murmur, the direction of its transmission, the arterial phenomena and the other features of aortic disease. *Endocardial murmurs* are not affected by position or pressure and rarely simulate the pericardial affection. *Pleuro-pericardial friction* is diagnosed by its disappearance when the breath is held; it has no to-and-fro character and is intensified during respiration.

Prognosis.—The prognosis is favorable so far as life is concerned, but adhesions may seriously complicate the condition, as the two layers of the pericardium may become agglutinated. This condition also may be the forerunner of the chronic form.

Treatment.—The patient should be kept absolutely quiet in bed. The *diet* should be bland and the amount of liquids should be limited to avoid over-distention of the vessels. Cardiac pain may call for hot compresses or possibly the icebag. The bowels should be emptied by enemas or carthartics if any constipation is present. In case of extreme cardiac excitement it will be necessary to use heart stimulants. For this purpose *strychnia* (gr. 1/60-1/30) is our best tonic and it should be given at least three or four times daily. Later *digitalis, adonis* or *convallaria* may be demanded.

The homeopathic remedies usually indicated at first are: *Aconite,* which covers the fever, dry surface, thirst and also the cardiac and mental excitement. It especially relates to the precordial pain and the sense of constriction. *Spigelia* is indicated more particularly when the symptoms assume an anginal character, and the pain radiates down the chest and the left arm. *Rhus tox* may be thought of in this respect, but there is an underlying toxic state. *Veratum viride* and *glonoin* should be used with the idea of inducing arteriole dilatation. *Bryonia* and *cantharis* will invariably relieve the excruciating pain in the primary stage. *Gelsemium* is also related to the deep seated muscular pains which attend the rheumatic form of invasion. *Colchicin, sodium salicylate, kalmia, gaultheria, potassium iodid,* and *argentum nitricum* will be of service in accordance with their indications. When the nervous symptoms are extreme it may be well to resort to the use of a sedative unless relief is obtained by indicated remedies.

SERO-FIBRINOUS PERICARDITIS.

This form of pericarditis, in which the effusion of serum into the pericardial sac is the characteristic feature, attends or follows the dry or plastic form. It is not to be presumed, however, that every case of dry pericarditis terminates with a liquid exudate. It is secondary to rheumatism, renal diseases and eruptive fevers. Pneumonia and tuberculosis are often predisposing causes and typhoid fever is possibly a causative factor. In fact, the causes are the same as in the plastic or dry variety.

Pathology.—Following the plastic exudation a second stage of secretion exists, in which there is a variable amount of effusion usually serous or serofibrinous in character in which pus or blood may be mingled. It begins as a fibrinous deposit on the visceral pericardium and as the serum and fibrin increase in amount they gravitate to the pendant portion of the sac. The *quantity* of serous effusion may vary from two to four ounces to two or three pints, in which case it pushes the heart upward. The *membrane* is thickened in proportion to the amount of fibrin in the exudate. In the *favorable cases* the serum is slowly absorbed, leaving the lymph to form adhesions between the visceral and parietal membranes which tend to obliterate the pericardial sac. Sometimes the serum is not absorbed and then a *chronic pericarditis* with effusion may persist for some time. If a *purulent exudate* is present, as in septicemia, puerperal fever, tuberculosis and nephritis, the visceral pericardium may be eroded and the cardiac muscle finally degenerate. Even in the ordinary case following rheumatic fever, there may be an inflammatory extension from the visceral layer into the heart structure. The *serofibrinous* variety does not, however, generally cause much myocardial degeneration. In protracted cases the muscular stroma may become softened, favoring atrophy or dilatation. When the fibrin element predominates, the tendency is toward cardiac hypertrophy.

Symptoms.—We are liable to overlook the symptoms of this form of pericarditis until the effusion has become pronounced. The signs of the acute attack may be clearly defined as before enumerated, yet as they subside we may be blinded by the often slowly developing exudate. *Precordial oppression* and pain, of varying character, will be experienced early. If the pleura is also affected, the pain may be severe. *Dyspnea* is a sign of increasing effusion, due to compression of the left lung and the right ventricle, thus disturbing the cardio-pulmonary circulation. For the same reason we shall find deficient blood aëration with feeble peripheral circulation. General *cyanosis* and *dropsy* may be present. From this mechanical cause, we shall invariably discover a small, feeble and irregular pulse. During inspiration the radial pulse may become quite small or disappear (pulsus paradoxus). When the effusion develops slowly the pulse does not show such marked changes, but when it is rapid and excessive the mechanical oppression is sufficient to cause pronounced alteration in the pulse. When plastic pericarditis exists the heart's action is accelerated, but as the fluid accumulates there is irregularity of rhythm, intermittency and often reduplication of the second sound. The *exudate pressure* may be so extreme that the esophagus and recurrent laryngeal nerves are pressed upon, causing difficult deglutition and aphonia.

Fever is not a prominent symptom; the temperature rarely reaches 103° F., though it may be ushered in by chilliness or rigors. The best observers have advised us to look out for pericarditis when, in rheumatic fever, there is an accession of fever without the extension of inflammation to additional joints. *Nervous symptoms* have been discovered during the course of this disease, which later may suggest melancholia or maniacal delirium. Headache and mild delirium are frequent symptoms and convulsions and coma may occur, particularly in young subjects.

Physical Signs.—*Inspection.*—The skin and mucous membranes are slightly cyanotic. The cervical veins are prominent and pulsate. The expression is anxious. The respirations are rapid, labored and often irregular. The patient lies in bed with the head and shoulders elevated, or may assume a sitting posture. *Precordial bulging* may be noticed in young patients and sometimes in cases with abundant exudation in older people. The diaphragm, depressed by the pericardial exudate, may carry down the left lobe of the liver. The *apex beat* is at first exaggerated in impulse and later is displaced upward and outward, becoming gradually weaker and more diffuse. When the pericardial sac becomes filled the apex beat may disappear. The respiratory expansion of the left side is frequently diminished and when the effusion is extreme there may be epigastric bulging.

Palpation.—By this we learn that the apex beat is diffused, feeble and displaced upward; its location may vary with the patient's position. A *friction fremitus* may be palpable at the cardiac base in the early stage of effusion or when absorption of the serum has taken place. *Fluctuation* can not plainly be detected. In large effusions the liver is easily palpated.

Percussion.—The chief revelation of percussion is the *increase* in the area of cardiac dulness. It gives the triangular outline representing the shape of the pericardium with base downward and the apex extending upward to the second interspace. Laterally the dulness is distinguished for one-half inch or more to the right of the sternum and more than twice the distance to the left. As the effusion continues the transverse area of dulness increases and may extend as far as the nipple on the right and to the axillary line on the left; the pericardial apex will then be pushed up as far as the first interspace, while the base will descend much below the transverse nipple line. It is possible that an emphysematous lung may to a great extent occlude the positive outlines of the pericardial effusion. Position will change the area of dulness. The presence of *vocal fremitus* will help to differentiate from pleuritic effusion. The margins of the lung surrounding the heart may be retracted and the heart carried forward or dilated, thus causing an increase in the area of dulness, which may not be due to the effusion.

Auscultation.—The *friction* sound which is characteristic of the dry form is heard only indistinctly at the base or apex. In the majority of these cases, however, this sound may be detected early, though it is feeble. If there is absorption of the liquid this sound will be restored unless there is abatement of the inflammatory feature. The *normal heart sounds* are gradually weakened, the aortic and pulmonary disappearing last. *Cardiac rhythm* is also perverted. In many cases broncho-vesicular breathing may be heard over the lower lobe of the left lung, on account of the compression at this point.

Course and Duration.—The course and duration of this disease will vary in accordance with the severity of the affection. In some cases the exudation is rapidly formed and excessive in quantity. In other cases the periods which define the dry, effusive and absorptive stages are short and atypical. The absorption of the fluid is often of long duration. Cardiac hypertrophy or dilatation, atrophy or degenerative changes may follow the chronic form. An *adherent pericardium* is a common sequela. The temperature disappears by lysis when recovery begins. *Fatal termination* is indicated by a persistent fever or sudden hyperpyrexia followed by marked dyspnea, cyanosis and failing circulation. Active delirium, restlessness, insomnia, subsultus and involuntary action of the sphincters presage a sudden sinking of vital forces.

Diagnosis.—The diagnosis of pericarditis is often difficult. The typical case, if watched throughout its developing stages, may be correctly diagnosed if the case is given careful study. The physician is often called when the disease has existed for some time and when various complications are liable to mask the characteristic features. In general we may say that the presence of the friction sound, the triangular area of pericardial dulness with possibly precordial bulging with circulatory disturbance as above mentioned ought to settle the diagnosis. We must differentiate the following diseases: *Endocarditis* will be recognized by a sound which is synchronous with the heart sounds; that is, they precede, follow or replace the systolic or diastolic sounds. Endocardial murmurs are more diffusible and extend to the left or right of the cardiac region; they are also transmitted along the arteries. The endocardial sound is less superficial than the pericardial and it is not affected by the patient's change of position.

Cardiac dilatation presents an impulse which, particularly in thin chested people, is visible, wavy and palpable, though feeble and is not accompanied by effusion. The heart sounds in dilatation are clear though weak, in contradistinction to the distant and muffled sounds in pericarditis. No perceptible fever or pain attends the typical dilatation, though there may be the history of a chronic valvular disease with all its concomitant symptoms. *Acute serofibrinous pleurisy* on the left side often simulates an attack of pericarditis and the two diseases may coexist. The pain of pleurisy is, however, more incisive; the percussion dulness extends lower and farther to the left; the pleuritic friction sound is heard lower and is synchronous with the respiratory movements; it ceases in the temporary suspension of respiration.

Prognosis.—We may say that the ordinary case of pericarditis with effusion terminates favorably, providing the general condition of the patient is not decidedly below normal and the virulence of the affection is not unusual. Cases associated with rheumatism, pneumonia and other tractable infections, yield generally to early and thorough treatment. Pus in the exudate always makes the case more unfavorable. Accompanying nephritis should always cause an unfavorable prognosis.

Treatment.—The treatment of pericarditis with effusion should look toward the subjugation of the inflammatory invasion, the regulation of cardiac activity, the relief of pain and distress and the removal of the exudate, if necessary, by surgical means. *Absolute rest* in bed and perfect quiet are also mandatory requirements. The regulation of cardiac action requires great caution from the fact that sudden depression is exceedingly dangerous. The coal tar derivatives should never be resorted to for this purpose. *The icebag* is still our safest method of sedation. In some cases we shall still find the hot application or possibly the almost obsolete poultice efficacious. The hot flannel, or warm cotton or wool applications may be better in other cases. *Opium* internally, or a poultice sprinkled with laudanum, is still a means for local relief. *Dovers' powder,* in five grain doses, *codein* or *morphin* are often essential internal remedies. Some specialists still advocate the use of leeches and, no doubt to advantage in certain cases. The same may be said of bleeding from the arm when the circulation is embarrassed and the pulse is irregular; it may be even more positively indicated when there is arterial anemia, together with venous congestion.

Stimulation is a subject demanding the most careful consideration when the pulse is weak and the cardiac power is persistently failing. *Strychnia* (gr. 1/60-1/30) hypodermatically administered is on the average the best agent for this purpose. The combined tablet of *digitalin, strychnia* and *nitroglycerin* may be needed in some cases. *Spartein sulphate* (gr. 1/10) is also a valuable remedy for this purpose. If the heart is embarrassed by the excessive effusion, it is not wise to resort to stimulation except after the removal of the exudate which is a mechanical cause of the trouble. *Paracentesis* is essentially a surgical procedure and should not be performed by anyone but a surgeon except in emergency. The indications for this are found when the heart is greatly embarrassed in its action, and grave dyspnea suggests a serious compression. The exploratory incision under aseptic precautions may be resorted to if the physical and objective signs are not sufficiently confirming. The fourth or fifth intercostal space, about an inch to the left of the sternal margin, is the place for this. The aspirating needle or trocar should be employed for the slow withdrawal of the serous or serofibrinous fluid. If this should be purulent a pericardial incision should be made in the same place, a soft drainage tube should be inserted and the cavity may be washed out with a warm normal salt or boric acid solution. This again should be done by a competent surgeon. There are some practicians who still hold to the idea of using a blister followed by hot poultices for the relief of large effusions. This may possibly be employed when other measures seem inadequate. *Mercurial inunctions* have been advocated for the slowly disappearing effusions. Much may be done to reduce the effusion by increasing elimination through the skin, kidneys and bowels. *Cascara* and the *compound lapactic pills,* which relieve the liver of its torpidity, are worthy of trial. *Calomel,* in half grain doses, is permissible in obstinate cases. The *diet* should be extremely simple and fluids should not be given in large amounts lest the effusion be increased. *Rest* is as imperative here as in any other cardiac affection.

The *internal medication* should apply to the predisposing disease, the cardiac perversion and the relief of the effusion. As the cause may be frequently traced to rheumatic fever we shall be obliged to give this line of treatment precedence. The salicylates have been employed almost exclusively in the past, but are supplemented now by many other remedies. Possibly in some obstinate cases with a rheumatic history, we may obtain relief from gradually increasing doses of *sodium salicylate.* This is best given after meals, beginning with five grain doses and increasing to fifteen or twenty; it should be given with plenty of water. *Colchicin* with *methyl salicylate* is a recent preparation having some advocates. The objection to any salicylate lies in the gastric disturbance, which often discounts all its supposed utility. *Potassium iodid* is still looked upon as a valuable remedy whenever specific taint or a doubtful dyscrasia underlies the disease manifestations. It is undoubtedly also efficient in reducing the effusion. *Strontium iodid* may be considered in the same category.

Colchicin is also a serviceable remedy. Goodno claims that it is "a valuable preventive of rheumatic pericarditis." A solution of one grain of Merck's colchicin to one ounce of alcohol is the preparation used. Three to five drops of this are given every three or four hours. The dose should be gradually increased to the point of gastro-intestinal disturbance. *Spigelia* is one of the most useful remedies from the very first indication of the disease; one of its common symptoms is a neuralgic pain in the cardiac region, which radiates down the arm and is attended with cardiac palpitation. *Aconite, bryonia, apis, arnica* and *veratum viride* would naturally be indicated in the early stage of effusion when the cardiac excitement is due to the first inflammatory invasion. These remedies, with the exception of aconite and apis, I have not found successful in any potency higher than the first decimal. *Cannabis sativa* will often relieve the precordial pain when it is of a violent, piercing character and the patient displays much mental excitement. *Cactus grand.* will also control many of the early symptoms when the rheumatic disorder causes unnatural heart action and there is the reaction of exhaustion to the extent that the heart feels oppressed as if by a heavy hand. The *cardiac perversion* will be regulated by the remedies naturally indicated in accordance with its physiological disturbance. *Digitalis, convallaria, kalmia, adonis vernalis, naja,* etc., will generally answer this purpose, though the support of *strychnia* may sometimes be needed.

The remedial relief of *the effusion* will call for prompt and persistent attention. The best diuretic action is obtained by one to two drachm doses of infusion of *digitalis* every three or four hours, and in severe cases I give equally often ten grain doses of *diuretin.* Hydragogin is even a better remedy than digitalis. The patient may take small quantities of distilled water daily and in some instances this may be given more freely. Effervescent alkalithia, in teaspoonful doses, may favor the increase of urine. *Apocynum* tincture (m. 10-20) or possibly the aqueous solution in larger doses, may also encourage diuresis. *Stigmata maydis* in twenty drop doses of the fluid extract will also help to obtain this result. In addition to this we should also resort to the regular use of high normal salt enemata and the hot bath at stated intervals. *Apis, arsenicum, cantharis* and *elaterium* are good remedies when indicated and will surely aid in reducing the effusion. As a standard remedy for this purpose we shall find frequent use for *potassium acetate* (grs. 10-15) every three or four hours. *Potassium iodid* is also serviceable. This should be given in increasing doses of the saturated solution.

PURULENT PERICARDITIS.

This disease has also been termed empyema of the pericardium. The cause is generally traced to some acute infectious disease, though it may follow the sero-fibrinous form.

Many times we find it associated with the tuberculous process, while the pneumococcus, gonococcus and pyogenic bacteria have also been found in the pus. The pericardial membrane is thickened and granular, while the myocardium may become softened and show fatty degeneration.

Symptoms.—In addition to the typical symptoms of pericarditis with effusion, we shall have the febrile signs which usually attend the development of pus. There will be *rigors* and *high temperature,* with cold sweats and pronounced prostration. The small, rapid and *irregular pulse* attended by the peculiar temperature curve of the suppurative type, will clearly define this condition. It runs a rapid course and the result is almost invariably unfavorable. The symptoms of the *primary cause* will generally be associated with the typical signs of suppuration.

Diagnosis.—The diagnosis should be confirmed by the symptoms mentioned above and the additional evidence obtained by exploratory incision or aspiration.

Prognosis.—It will be necessary to give an unfavorable prognosis in every case of this kind.

Treatment.—This is essentially a surgical disease, as drainage of the pericardium is necessary. The general treatment will be similar to that of serofibrinous pericarditis. Such internal remedies as *hepar sulphur, arsenicum iodid, sulphur, iodoform* and *mercurius* may be used with some degree of success. One to three grain doses of *methylene blue* may also be given. *Boric acid, benzosol* and *beta naphtol* may be considered for their antiseptic action.

ADHESIVE OR CHRONIC PERICARDITIS.

It has been supposed by some that this form of pericarditis exists independently of the acute form, inasmuch as it is sometimes very difficult to discover any preceding acute symptoms. That it succeeds the acute variety there is hardly any doubt. It may follow the plastic form or it may appear after the absorption of the effusion. The adhesions may consist only of a few fibrinous bands, or the opposing membranous surfaces may be firmly adherent for their entire surfaces or in limited areas. Often the result may be a completely obliterated sac, due to an excessive thickening of the layers. Calcareous development may be present or attendant valvular lesions may create marked hypertrophy of the heart. The myocardium, too, may suffer a considerable degeneration. The complete adhesions occur most frequently in children. Chronic tuberculous pericarditis may precede or follow this condition.

Symptoms.—We do not often find characteristic subjective symptoms, except as they relate to the primary cause. Functional weakness will be recognized early and if there is an associate valvular lesion, we shall discover an apical murmur. Degenerative changes in the heart muscle may be in evidence. Venous stasis, dropsy and dyspnea may reveal the weakness of the right ventricle, while the pulse will be rapid, irregular and of low tension. The pulsus paradoxus may be noted.

Physical Signs.—*Inspection* may show a depression or pitting in the intercostal spaces over the heart or a retraction of the chest wall over the apex, synchronous with the heart's systole, may be noticed. This is more pronounced during inspiration, and may be detected also by palpation. We must remember that apical retractions may be detected in connection with other diseases of the chest. A diastolic shock my be found which is due to the cardiac rebound during diastole, and is considered a more positive sign. The collapse of the jugulars during diastole (Friedreich's sign) is a physical phenomenon of some value, though this may be observed in cardiac dilatation without adhesions. The fixed position of the apex beat when the patient is turned on the left side is a valuable diagnostic sign. The character of the cardiac impulse is sometimes wavy and undulatory.

Percussion shows an increase in the area of cardiac dulness, especially upward and to the left. On account of adhesions between the pleura and pericardium this area of dulness may be fixed and uninfluenced by deep inspiration. *Auscultation* does not give any valuable diagnostic signs that are peculiarly distinctive. There is sometimes heard a group of loud murmurs caused by the "jogging" cardiac action. Other murmurs will relate to the complicating valvular lesion. The physical signs of hypertrophy, dilatation and myocarditis are to be discovered and differentiated whenever they may appear.

Prognosis.—This will depend upon the extent of the adhesions, the age and the physical condition of the patient, and the degree of structural change which may develop in the cardiac muscle.

Treatment.—We can not expect to accomplish much by treatment except through the symptomatic prescribing of. remedies for the valvular conditions which may arise. This is simply a mechanical difficulty and except for the general care of the patient we can do but little.

HYDROPERICARDIUM.

This condition has also been termed a dropsy of the pericardium, inasmuch as a serous transudation is found in the pericardium without any inflammatory invasion of the pericardial membrane.

Etiology.—Hydropericardium is frequently associated with general cardiac or renal dropsy. It is observed in connection with advanced chronic nephritis, in which cases it may appear suddenly. It is also a post-scarlatinal complication, though it may follow any infection. Hydrothorax may precede it. Mechanical pressure from mediastinal tumors, aneurysm or thrombosis may be a predisposing factor.

Pathology.—There is no evidence of pericardial inflammation. There may be a slight pericardial thickening due to interstitial edema. The effusion is light yellow in color with a slight amount of albumin and no fibrin. There may be an admixture of blood when the capillary vessels are impaired. The lungs may be compressed when the effusion is large. When the exudate is milky the condition is termed chlyopericardium.

Symptoms.—*Dyspnea* is a characteristic symptom due to the effusion which is often greater than is found in pericarditis. The *area of percussion dulness* is increased and varies with the patient's position. *Friction sounds* are absent. The *constitutional symptoms* are related to the causative cardiac or renal disease.

Diagnosis and Prognosis.—The history of the case and the symptoms defined above should help in making the diagnosis. The *prognosis* will depend upon the cause, the complications and the extent of the effusion.

Treatment.—The case may be treated similarly to those of pericarditis with effusion. Paracentesis may be performed, sometimes with favorable results. The following remedies may be used for internal medication: *Sulphur, silicea, calcarea fluorica, arsenic, apis, lachesis, digitalis, apocynum and strophanthus.*

HEMOPERICARDIUM.

Hemorrhage into the pericardium is a rare disease. It may result from injuries, such as gunshot wounds and stabs, or from rupture of the heart, dependent on cardiac aneurysms or fatty degenerations; again it may come from perforation of the sac by aneurysms of the aorta and coronary arteries. The aneurysmal rupture is rapidly fatal, while rupture of the heart muscle may not result in death quite so soon. The *symptoms* depend much upon the rapidity with which the blood escapes into the pericardium. The symptoms of effusion, with marked dyspnea and cardiac failure, appear very early in a majority of the cases. Pericarditis may follow if death is delayed.

The *diagnosis* before death is a difficult matter, except as we may know by previous examination that an aneurysm or degenerative condition existed. Sudden pain, dyspnea and failure of circulation, together with a rapidly increasing area of dulness, will suggest this disease.

The *prognosis* and *treatment* will give but little hope. Some help may come from remedies, but this will only be temporary. Aspiration has been successfully performed, but rarely gives relief.

PNEUMOPERICARDIUM.

The presence of air in the pericardium is a rare affection. Pus is also found in conjunction with the air, hence the term pyopneumopericardium is properly used in this case. We may look for the causes in the existence of wounds or in fistulous connection between the lungs and pericardium; tuberculosis and empyema may be factors in this respect. The decomposition of liquid pericardial effusions has been urged as a possible cause. Abscess in the liver or mediastinum or ulcerations from malignant growths may also be the cause of pneumopericardium.

Symptoms.—The symptoms simulate those of acute pericarditis with effusion, but the dyspnea is more severe. The *physical signs* are distinctive; the precordia is distended; tympanitic percussion-resonance is distinguished in the precordial region. *Auscultation* discovers a peculiar, splashing, metallic sound, caused by the movements of the heart in the fluid contents of the pericardium. These sounds may be heard at some distance from the patient. The heart's action is very weak and is exaggerated by the sitting position. The *prognosis* is grave and treatment yields little relief.

ENDOCARDITIS.

By this term we mean an inflammation of the lining membrane of the heart. As this is usually confined to the valves, it is often defined as a valvular endocarditis. It occurs either in the acute or chronic form. Under the first head we recognize (a) simple acute endocarditis and (b) ulcerative or malignant endocarditis. The chronic form represents an interstitial endocarditis of slow formation in which we shall find a sclerotic change that affects the valves most decidedly.

SIMPLE ACUTE ENDOCARDITIS.

Etiology.—There is no doubt of a bacteriological origin in most cases of endocarditis. While the micro-organism can not easily be found in certain cases, we still have reason to believe that infection is the cause of most diseases of this kind. Therefore, it is essentially a secondary disease. *Acute rheumatic fever* is its most prolific cause, many writers claiming it is present in sixty to eighty per cent of the cases. The young are particularly predisposed after even a slight attack of rheumatic fever. Endocarditis may be the chief symptom of rheumatic fever when the articular signs are not in evidence. We will, however, observe the most characteristic cases when the articular involvement is severe. The relation to tonsillitis and chorea will tend to confirm the association of these diseases with rheumatism. All *specific fevers* probably predispose to endocarditis, though we shall discover that it is associated with scarlet fever more frequently than with typhoid, diphtheria, measles, variola and varicella. *Gonorrheal rheumatism* may predispose to it, and sometimes it is secondary to syphilis. *Pneumonia* and *pulmonary tuberculosis* contribute a small proportion of cases. *Parenchymatous nephritis,* diabetes, gout, ulcerative carcinoma and the suppurative diseases furnish occasional causes. *General debility,* the pregnant and puerperal state are regarded as possible sources of endocardial infection. *Injuries* to the valves and the chordae tendineae, as well as inflammatory extension, may be regarded as rare causes.

A form known as *acute recurrent endocarditis* may follow a preexisting sclerotic endocarditis. *Men* are supposed to suffer with this disease more frequently than women, and in this respect we may state that alcoholism probably accounts to a great extent for this.

Pathology.—The pathological process is characterized by vegetations in the form of excrescences varying from a small point to a pea in size or even larger. They are found chiefly on the valvular surfaces near the margins and opposing the blood current. They often form a row of "beadlike outgrowths" and may involve the tissues surrounding the valves. Sometimes the chordae tendineae are alone affected. The *mitral segments* are most frequently involved and the vegetations will be found on the auricular side. Should the aortic valves be involved, we shall find the vegetations on the ventricular surface. These abnormalities may be merely an elevation of morbid tissue, or the vegetations may be attached by pedicles which, being easily detached, predispose to embolism of some distant blood vessel. Should endocarditis be a possibility during fetal life, the tricuspid valves would more naturally furnish a location, as the right heart is then the active part.

In the *mild form* the valves are simply swollen and smooth, and finally the vegetation is absorbed or a small indurated mass remains. On the area of infiltration a layer of fibrin is often deposited which, being easily separated, may cause further inflammatory trouble either in the heart or distant blood vessels. The new growth may degenerate and undergo calcification. The morbid process may invade the cardiac walls or the cell proliferation may terminate in the ulcerative variety. The more common termination is the chronic or sclerotic valvulitis. The friction caused by the blood current against the roughened valves aids the inflammatory development caused by the micro-organisms and their toxins.

The *pathogenic factors* which develop the disease are claimed to be the staphylococcus pyogenes aureus, diplococcus pneumoniae, the streptococcus pyogenes and sometimes the bacillus coli communis, the gonococcus, the bacillus diphtheriae, the bacillus of Eberth and possibly other micro-organisms.

Symptoms.—*Definite symptoms* are not always obtained and many times early suggestions of the disease are obscure until the inflammatory invasion affects the cardiac walls. In an attack of articular rheumatism or any other infectious disease the heart should be examined daily to detect any beginning endocarditis. *Precordial pain,* dyspnea and cardiac palpitation with moderate fever (101°-103° F.) are the early characteristic symptoms. The pain is not always sharp or lancinating; usually it is only a dull precordial distress. With children it may be referred to the epigastrium. The *dyspnea* may be paroxysmal or constant. The labored breathing may be attended by impaired circulation. The *pulse* is generally excited at first, but later becomes weak and irregular. Cardiac enfeeblement, with arrhythmia, is a supplementary suggestion of the disease. A rapid rise of temperature is a confirming sign.

Physical Signs.—*Inspection.*—The area of visible impulse may be increased, though usually it is normal. As the heart becomes weaker the pulsations are less distinct. *Cyanosis* and distention of the vessels of the neck are rare symptoms. *Palpation.*—The endocardial thrill may be felt by the hand. The increased impulse of the early stage and the lessened intensity of the later stage may also be frequently detected. Recurrent endocarditis associated with previous hypertrophy, may present a heaving apical impulse. With acute dilatation this may be greatly weakened. *Percussion* will reveal a normal area of dulness, though this may be increased transversely when dilatation occurs. *Auscultation* discloses the most characteristic feature of endocarditis. This is the soft blowing *systolic murmur* heard best at the apex, because the mitral valves are generally the seat of the lesion. In the early stages there is little transmission of the sound as the inflammatory changes are not thoroughly established; this may develop later. The intensity increases with the progress of the disease. The murmur is caused by the insufficiency of the mitral valves or the roughened endocardium of the ventricle. Prolongation of the first cardiac sound is another confirming sign. Aortic accentuation and later pulmonary accentuation may be recognized. An aortic murmur sometimes coexists, and a relative incompetency of the tricuspids may give rise to a short and low-toned murmur over the tricuspid orifice. In rheumatic cases these murmurs may become very harsh.

Diagnosis.—If the signs mentioned above are found in the physical examination a diagnosis should be arrived at without much doubt. *Acute febrile diseases* may yield a similar murmur, but these will be heard best over the aortic area, while the endocardial murmur is located over the mitral area and at the apex. In *pericarditis* we shall not be able to easily settle the differentiation on account of the confusing pericardial friction sound and the subsequent presence of effusion. When, however, the pericardial and endocardial conditions co-exist, the endocardial murmur will be distinguished first. The friction-sound in pericarditis is more superficial and is affected by postural change and local pressure. The murmur established in conjunction with a gradual hypertrophy following a *neurotic cause* will be differentiated by the preceding tachycardia and the symptoms which define the causative disease. The *relative mitral murmur,* which follows an aortic lesion or a protracted disease like anemia, will be louder and less uniform in character; in this case there will be only a slight accentuation of the pulmonary second sound and a small and irregular pulse. The *functional murmurs* are generally heard best at the cardiac base.

Prognosis.—The danger in endocarditis depends greatly upon the primary disease. Without complicating conditions the ordinary case should respond to prompt and careful treatment. The development of a permanent mitral lesion is the greatest danger. Embolism, albuminuria and hemiplegia are liable to follow an endocardial attack.

Treatment.—*Rest* is a most essential measure. The greatest danger in the early stages of the disease comes from overexertion and excitement. The *diet* should be nourishing, but light. Nitrogenous food is preferred and an excess of liquids should be interdicted. Protection against taking cold should always be advised. Many practicians claim that the patient should be kept between woolen blankets, but it is often possible to maintain warmth without this irritating covering. To some wool is decidedly unpleasant. The *warm bath* is always a healthy cardiac sedative, but we should be on guard against its depressing tendency. Some light *stimulant* may be needed following this. Systematic regular sponging may be more beneficial, as it is not attended with so much nervous excitement. The *nervous system* should always be watched carefully. The early treatment of the primary disease is important. *Rheumatic fever* (the treatment of which has already been outlined) will demand much attention in this respect. For precordial pain we should not forget the hot application or possibly the icebag.

During the early symptoms we shall be obliged to relieve the pain and the nervous excitement. *Bryonia* or *spigelia* will generally suffice in this respect, though *cimicifuga* and *rhus tox* should not be neglected. *Spigelia* should naturally be preceded by *aconite*. *Rhus tox,* I believe, is too frequently neglected in these cases; its relation to rheumatic and toxic conditions should lead to its use. Besides, the restlessness and general debility, accompanied by relief from motion, are particularly suggestive. The tendency to erysipelatous inflammations and the swelling of the extremities which are characteristic of this remedy represent the low febrile condition which so often prevails.

Arnica, rhododendron and *ranunculus* should be compared for their distinctive symptoms. After *rhus tox* we shall often observe the usefulness of *gelsemium,* which pertains to a more decided general muscular weakness with chills, pain and paralytic prostration.

Laurocerasus will be indicated for the spasmodic throat and chest contractions, and also for the suffocative attacks which are in evidence during great nervous excitement or when dyspnea is pronounced. These symptoms will be associated with some pain in the precordial region, while cyanosis is liable to follow. *Kalmia* displays similar symptoms, but there is more pain in the cardiac region. *Cactus* will evince more weight in connection with the pain, the patient often complaining of the "heavy hand" over the heart. *Kali carb.* comes under this category, only it will be called for when tissue depletion is more complete and there is frequent cardiac palpitation because of it.

Stimulation of the heart may be necessary, though this should be avoided as much as possible, particularly if there is myocardial degeneration. *Digitalis, convallaria, strychnia, nitroglycerine* and *spartein,* together with other remedies previously mentioned, must be considered here. Whiskey and the aromatic spirits of ammonia may be temporarily sufficient for this purpose. *Ammonium carb.,* in frequently repeated doses, may be sufficient in this respect. The violent action of the heart will be easily regulated by *veratrum viride,* and while this remedy should be given in tincture doses, it should not be pushed too far. I use twenty to thirty drops in one-half glass of water, two teaspoonsful every hour. For the nervous manifestations which spend their force directly upon the heart, I know of no better remedies than *agaricin* and *ammonium val.,* in the second decimal potency.

As the *acute symptoms subside* we shall obtain good results in restoring the heart's function by the use of remedies which tend to encourage absorption or elimination of the pathologic exudate. Here we may resort to the use of *kali iodid, iodin, spongia, arsenicum iodid, baryta iodid* and *strontium iodid. Aurum chlorid, mercurius, sulphur, lachesis,* and the *calcareas* may be used under similar conditions. The aim should be to protect the valves and the myocardium from any further destruction or perversion and, for this purpose it will often be necessary to study carefully the pathologic status and the clinical symptoms. More than this, we must carefully individualize and diagnose the peculiarities of the patient's physical makeup, for these are truly important in the management of the case.

ULCERATIVE ENDOCARDITIS.

Various definitions have been given to this particular affection of the endocardium. Malignant endocarditis, acute septic endocarditis and diphtheritic endocarditis are the terms which have been used interchangeably. It is an extreme form of endocardial inflammation due to infection, and is characterized by its tendency to terminate in perforative ulceration and disorganization of cardiac tissue.

Etiology.—The disease is always a secondary one and while it is caused by a definite micro-organism, we shall invariably find that the symptoms and the lesions of the acute form precede its development. The streptococcus pyogenes is probably the specific irritant. The bacillus diphtheriae, the staphylococcus, the bacillus coli communis, the bacillus anthracis, the pneumococcus, the gonococcus and other organisms must also be recognized as possible causes. Typhoid fever, scarlet fever, erysipelas, chorea, tuberculosis and Bright's disease may furnish the infective material which enters a healthy heart by way of the blood current and attacks the valves or endocardium. The valves may become infected when they are in a healthy condition or the disease process may be encouraged by an existing valvulitis. Age and sex are predisposing factors. On account of the puerperal state we may discover it oftener in women. Middle life and childhood furnish the most cases.

Pathology.—The lesion will be found most frequently on the auricular surface of the mitral valves or on the ventricular surface of the aortic valves. This, of course, is due to the fact that the primary endocardial inflammation which predisposes has already affected that part of the viscus. In the early stages vegetations form, which later undergo necrosis. *Ulceration* is a common result of this inflammation; if it is superficial, it involves the endocardium and the valves; if deep, it affects the cardiac walls and septum, causing resulting perforation in some cases. Sometimes *abscesses* form at the base of these vegetations and these may rupture and produce extensive suppurative lesions and systemic sepsis. As the blood enters the abscess cavity it will cause the endocardium to pouch out, forming an aneurysmal dilatation of the valve. If there is perforation of the valve or the wall, the extravasation may be more extensive and serious. The *vegetations* may be detached in small or large masses and convey the infective material to distant organs or cause dangerous embolisms. The spleen, kidneys and brain often suffer from this infection. In the same way the serous cavities are liable to a similar septic invasion.

Symptomatology.—We shall not find the typical symptoms of the simple variety when this stage of infection is reached or when the suppurative factor is the cause. Often the early signs are confusing. A marked *intensity* of the inflammatory process, constitutional infection and embolic complications will characterize the true symptomatology. *Subjective symptoms* often pertain to other organs than the heart, while the local symptoms may be entirely absent, or be noticed only as a slight precordial distress. *Various types* have been accepted as representative of the different varieties of the symptomatology. For instance, a *typhoid variety,* not, however, exactly similar to typhoid fever is recognized by the initial onset with chills and rigors, which are repeated at intervals for several days. An irregular fever follows and there is progressive prostration, sweating, emaciation, marked nervous disturbances, gastro-intestinal irritation with tympanites, diarrhea and eruptions simulating the rose spots of typhoid. Delirium and coma may also be present. A cardiac murmur may or may not be found.

A *pyemic* group of cases forms another class. Here the infection of the primary disease is characteristic. Chills, fever and sweats are pronounced, and in many respects the features of malaria are in evidence. Progressive anemia will be noticed and the general depression is extreme. A valvular lesion may be detected or it may be masked by the pronounced systemic symptoms. Dyspnea, palpitation and cyanosis are sometimes present.

A *cardiac* type indicates a recurrent malignant endocarditis. This means a new endocarditis established on the lesion of a previous one. The secondary infection comes from streptococci or other organisms. The *onset* is abrupt and distinguished by a chill; the temperature may reach 104° F. or higher, and will show an irregular or intermittent curve. The old murmur may become louder and more blowing in character, though in some instances this can not be appreciated. The signs of failing compensation may be suddenly manifested and dyspnea, cyanosis and dropsy may bring on a sudden collapse. Sometimes we find a case with similar symptoms running a subacute course.

Cerebral cases constitute another variety under this head. Here the features of suppurative meningitis will be presented. The symptoms will point almost entirely to the brain affection without many indications of a cardiac lesion or a primary infectious cause. In fact, all varieties of infectious endocarditis will furnish more symptoms referable to remote organs and general sepsis than to endocardial ulceration. *Infarctions* of the brain, spleen, kidney, liver, lungs and other organs will present signs of local involvement, together with symptoms of systemic infection. The *temperature* will show periods of hyperpyrexia, followed often by sudden apyrexia. The cardiac signs will frequently be negative.

Physical Signs.—The physical examination will not help us so much as the proper interpretation of the symptoms. The physical signs are often entirely negative so far as the heart is concerned. A systolic murmur may be detected if it is not masked by other symptoms. The second sound is sometimes accentuated.

Diagnosis.—It is not an easy matter to recognize malignant endocarditis, unless we give a painstaking study to all the predisposing causes, as well as to the symptomatology. If we can find a chronic valvular lesion in addition to the general symptoms outlined above, we may be reasonably sure of our diagnosis of malignant endocarditis. The supplementary signs of embolism will be of value. It is very difficult to distinguish between this disease and pyemia. *Malaria* is established positively only by a blood analysis. *Typhoid fever* will be the most difficult to differentiate. *Septic endocarditis,* however, has a rapid onset and is marked by a chill and high fever, which has no regular temperature curve. Constitutional symptoms generally precede and are more severe. Precordial pain and sometimes a systolic murmur show the cardiac involvement. The profound prostration which begins early is different from the gradual emaciation of typhoid. In *malignant endocarditis* the blood shows the presence of a septic leukocytosis, while typhoid reveals a decrease in the number of leukocytes and the typical Widal reaction.

Prognosis.—The typical cases generally terminate in death. Usually this occurs after the first two or three weeks. The cases which recover are at present regarded as benign in character. The cases met in association with old cardiac lesions may respond to careful treatment.

Treatment.—The principal aim in the treatment will be to nourish the patient as much as the febrile condition will permit and to give remedies which will meet the septic condition. *Arsenicum alb.* will be thought of first, as it represents the asthenic character of the disease. It is best given in the third decimal potency. This may also be given in the form of Fowler's solution, or, better, in combination with iron and strychnia. *Lachesis* and *crotalus* should be considered in this group. *Quinin* and *chinoidin,* as well as the *arsenate of china,* together with *agaricin, eupatorium* and *cedron,* may be prescribed for the adynamic state, simulating malarial fever with its chills and copious sweats. *Baptisia, gelsemium, bryonia, echinacea* and *rhus tox* will conform to the typhoid symptoms, I should also use *methylene blue, salol* or *benzosol,* the same as in typhoid conditions.

CHRONIC ENDOCARDITIS.

This is a sclerosis which affects the valves and leads to a deformity of the segments on account of the contractions. It is an insidious process which may be a sequela of the acute form or possibly it may start as a subacute inflammation.

Etiology.—*Acute* and *chronic* rheumatism doubtless play a large part in the causation of this disease. Some cases, however, may originate from a very mild form of valvulitis which is often overlooked until the chronic condition is established. The *other infectious diseases* contribute their share to the chronic variety, but rheumatism exceeds all other causes, particularly in children. *Prolonged* or *excessive muscular exertion* is also a factor in the early cardiac debility which leads to this chronic state. The slowly developing interstitial change may also depend upon syphilis, malaria or other causes for its start. *Chemical irritants,* such as uric acid, alcohol, lead, etc., may be included under this head and mechanical influences may have something to do with the initial cause. *Arterio-sclerosis, chronic nephritis,* and even trauma, may be looked upon as conditions which lead up to this pathologic state. Predisposing encouragement may lie in a hereditary depreciation, or a malformation. *Age* and *sex* may, in certain instances, have something to do with the creation of a physiologic perversion which ends in chronic valvulitis.

Pathology.—The pathologic change will be noticed in the endocardium covering the valve alone or in the endocardium of the wall. Usually it is in the valve. The edges and bases of the segments may alone be affected or the free border of the leaflets may present a thickened appearance or small nodules. The normal elasticity is diminished and the leaflets have a tendency to shrink and curl up. The exudate may cause an agglutination of the segments and fibrin may be deposited in considerable quantity, which naturally favors induration of the parts involved. As the inflammatory process continues the connective tissue increase becomes more manifest. With the subsequent induration and contraction, and in some cases calcification, we shall observe a greater tendency to separation of the leaflets so that normal approximation is impossible. As the valves fail to close on this account, insufficiency naturally follows.

When the *sclerotic perversion* invades the aortic segments, obstruction to the outflow is liable to occur. *Fatty* or *calcareous changes* usually follow this morbid condition in the neighborhood of the aortic opening. Sometimes it spreads to the ascending aorta and the coronary arteries. This tends to prove that general atheroma may follow this disease. These sclerotic and atheromatous degenerations may in like manner affect the pulmonary orifice and the pulmonary artery. As the left auriculo-ventricular opening and the mitral leaflets suffer from the same sclerotic tendency, we shall find that stenosis of all the cardiac orifices, together with contraction of the valves which protect them, are liable to exist as sequelae of chronic endocarditis. When the free edges of the valves adhere, the contracting chordae tendineae draw the leaflets toward the apex, forming what is known as the hollow cone, or the united parts may be pierced by a narrow opening called the button-hole slip. *Ulceration* may also follow the atheromatous degeneration. Besides these pathologic changes, we may observe a chronic myocarditis, or embolism affecting distant organs. The perversion of the heart muscle as a whole and the separate chambers as well, will be considered under the special lesions of the heart. Inasmuch as the diagnosis, prognosis and treatment pertain to the valvular diseases which are to be found in another chapter, we discuss the treatment there.

MITRAL INSUFFICIENCY.

Under this head we shall study one of the most common of the valvular cardiac diseases. It has been defined as a regurgitation and incompetency, both of which terms refer to imperfect approximation of the valves on account of rupture or contraction of the segments and the consequent impaired flow of blood through the left auriculo-ventricular opening.

Etiology and Pathology.—The predisposing factors are age, sex, occupation, habits and previous diseases. It is observed more often in young adult males, probably because of their more arduous and strenuous occupations, and among those who habitually use alcoholic beverages. Arteriosclerosis, endocarditis (rheumatic and infective), pulmonary disease, aneurysms, aortic stenosis and insufficiency are also predisposing factors. Congenital deformities of the cardiac structures may favor its development, as will any disease affecting the chordae tendineae, columnae carneae or the myocardium.

Because of the various etiologic factors the following pathologic changes may be noted:

(a) Thickening of the valve-segments, especially along the margin, with the formation of vegetations or calcareous deposits.

(b) Adhesions between the valve-leaflets and the cardiac wall.

(c) Cicatricial contraction of the valve-segments, especially after ulcerative endocarditis.

(d) Atheromatous degeneration of the valves—a part of a general arteriosclerosis.

(e) Normal valve-segment with a dilated auriculo-ventricular ring, secondary or aortic stenosis or insufficiency.

(f) Disease of the chordae tendineae or columnae carneae, allowing prolapse of the valves into the auricle, or by shortening, preventing normal approximation.

(g) Dilatation and hypertrophy of the auricular and ventricular walls.

Certain physiological changes take place in the cardiac cycle as a result of the structural changes. At each cardiac systole the blood regurgitating from the left ventricle into the left auricle meets the blood coming from the pulmonary veins, and causes over-distention of the left auricle, which may result in dilatation of that chamber and hypertrophy of its walls. Likewise there will be increased back-pressure in the pulmonary circulation, resulting in over-distention and a compensating hypertrophy of the right ventricle. Should this compensation fail, there will appear a tricupsid insufficiency with right auricular dilatation, manifested by a murmur heard over the tricuspid valve and evidenced by general venous dilatation.

Thus we observe that the misfortune of mitral insufficiency is mitigated by hypertrophy and dilatation of the right heart. As this causes less blood to enter the aorta, it explains the presence of a longstanding low arterial tension.

Symptoms.—The symptoms of mitral insufficiency depend greatly upon the general health of the patient and the age and degree of the lesion. If the *predisposing* cause has not made unfavorable inroads upon the physical state and if a proper care is observed, there may be only slight symptoms suggestive of this disease. *Regurgitation* often takes place so gradually and the hypertrophy is so slow in its progress that no marked signs of the disease may be detected by casual observation. As the leakage increases and the pathologic developments appear we shall be able to discover something typical of the disease. The steady compensation of the muscular fibres tends to hold the heart quite near to its normal action, unless some unusual strain is put upon it. We therefore describe the symptoms in relation to this process of hypertrophy. When *compensation* begins to fail we may notice symptoms indicative of pulmonary congestion. There may be nosebleed, hemoptysis and evidence of edema of the lungs. *Dyspnea,* a slight cough, expectoration of frothy or bloodstained serum and palpitation are additional symptoms. All of these signs will be augmented by excitement and over-exertion. *Anasarca* is generally slight during this period, but in exceptional cases it may be quite extensive. *Gastro-intestinal* irritation is a common complaint. As the *regurgitation increases* and the left ventricle becomes weaker, the aorta will receive less blood and as a consequence the pulse will be small in volume. *Cyanosis,* cold hands and feet and a pinched facial expression are significant of this stage. Besides this we shall find an undue prominence of the facial vessels and in patients with congenital insufficiency there may be more or less clubbing of the finger ends.

When *compensation fails* completely we shall find evidence of dilatation. The *first symptom* of prominence is cardiac weakness. The *pulse* becomes irregular, *palpitation* and dyspnea increase and the signs of pulmonary congestion are augmented. The *general venous engorgement* is marked and this will be shown not alone by peripheral stasis, but also by the enlargement of the liver and a catarrhal inflammation of the intestines. *Hepatitis,* with acute catarrhal jaundice, hemorrhoids and other signs of portal congestion are in evidence. The *urine* becomes scanty and often contains an abundance of albumin, many tube casts and blood corpuscles. *Edema* beginning in the feet extends upward until general dropsy is present.

Physical Signs—*Inspection and Palpation.*—The area of visible impulse and palpable percussion is greatly increased and the apex beat is *displaced downward* and *outward* in proportion to the degree of hypertrophy. In children the precordium may bulge. *When compensation is good* the cardiac impulse is heaving and forcible; when it fails it is weak, wavy and diffuse. A *systolic thrill,* synchronous with the first sound, is often felt at the apex. *Epigastric pulsation* will indicate right ventricular hypertrophy with dilatation, while *pulsating jugulars* and possibly a *pulsating liver* may suggest a tricuspid incompetency. The *pulse* during the early stages may be fairly full and regular, but as compensation fails it becomes small and compressible; as the heart failure increases it will be exceedingly irregular.

Percussion.—Cardiac dulness is increased transversely in proportion to the degree of hypertrophy and dilatation which affects the right and the left heart.

Auscultation.—A blowing systolic murmur—with and following the first sound—will be heard with greatest intensity at the apex. This may at times be heard best at the base or along the left sternal edge. It may occasionally disappear during the erect posture and reappear while the patient is lying down. The *sound is transmitted* toward the axilla and may be heard posteriorly at the angle of the scapula, unless there is fairly good compensation when it will be confined to the neighborhood of the apex. A *loud sound* will indicate a healthy hypertrophy. The *pulmonic second sound* is accentuated on account of the increased tension in the pulmonary vessels when the right ventricle is involved. A *combination of murmurs* will be detected in extreme cases. A *relative* soft and low-pitched systolic murmur may be heard at the base of the ensiform cartilage when the right ventricle is dilated. A presystolic murmur is, in rare cases, associated with the typical mitral murmur.

Diagnosis.—It will be necessary in making a diagnosis to distinguish the characteristic systolic murmur which is transmitted to the left and heard also posteriorly. A decided accentuation of the second pulmonic sound will be found in the majority of cases and there must be some evidence of right and left heart involvement.

A *differential diagnosis* must be made between this and other murmurs which will be discussed under separate heads. Here we must, however, distinguish between this and the systolic murmur of aortic stenosis and a similar murmur heard in association with functional diseases. In *aortic stenosis,* the murmur coming before the second diastolic sound will naturally prolong anl simulate the systolic sound; but this will be detected more clearly at the aortic interspace and along the blood vessels of the neck. We shall also notice in this an *absence* of the accentuation of the second pulmonic sound. In aortic stenosis we also find only an enlargement of the left ventricle. The *thrill* of *aortic stenosis* is also felt best at the cardiac base at the right of the sternum. Other differentiating points will be observed in the further study of this lesion. In the *functional murmurs* which simulate that of mitral insufficiency, we shall find evidence of anemia, general debility or some neurotic cause. Rheumatism is not a factor here. The skin shows a pallor instead of the dusky color of mitral insufficiency. The impulse is not so vigorous; the arterial pressure is increased in place of the forcible impulse and lowered pulse tension of the true mitral involvement. The apex pulsation is not transmitted toward the axilla and the soft systolic murmur is associated with a similar basic systolic sound and a venous hum in the neck.

The *relative mitral murmur* will generally occur in patients past middle life and in association with gout, syphilis or alcoholism. Arteriosclerosis will also be a factor in such cases; we shall also invariably find a diseased condition of the aortic valves. If a mitral systolic murmur is heard with an attending accentuation of the aortic second sound, particularly following renal disease with symptoms of high arterial tension, we may define it as a relative mitral insufficiency.

Prognosis.—The outcome of this disease depends entirely upon the predisposing causes, the age, the general health of the patient and the degree of compensating hypertrophy. *Progressive cardiac weakness* and sudden dilatation are liable to lead to grave results. The early and persistent treatment should insure fairly favorable results in the average case. The points in treatment will be discussed under valvular diseases.

MITRAL STENOSIS.

This is a condition of obstruction to the passage of blood through the left auriculo-ventricular opening. The constriction of the orifice results usually from endocarditis of rheumatic origin. Adhesions of the free borders of the valves or of the chordae tendineae are rare causes. It is observed mostly during early life, which may tend to prove the frequency of rheumatism or chorea and chlorosis as predisposing factors. Measles, scarlatina and whooping cough have developed it in children, while arteriosclerosis and chronic nephritis have predisposed in older subjects. Females are affected more than males. It is not so common as mitral insufficiency.

Pathology.—This pertains to the valves, the chordae tendineae and the structure of the ring, so that the orifice is reduced in calibre. The *constriction* may be due to a slight roughness of the surfaces, or rigidity of the valves. On the other hand it may be increased by *inflammatory* and *calcareous deposits* to such an extent that only a small amount of blood can pass through the opening. The "funnel shaped" form of stenosis, due to the adhesion of the valvular margins, or the "buttonhole slit" may occlude a greater portion of the orifice.

The *mechanical changes* which follow in the wake of this pathology are observed first in the dilatation and subsequent hypertrophy of the left auricle. On account of the obstruction to the passage of blood through the left auriculo-ventricular opening, the left auricle dilates and is compensated by muscular hypertrophy. Inasmuch as the walls are naturally thin this protection is limited. As the blood pressure in the pulmonary circulation increases, the right ventricle dilates and its walls are correspondingly hypertrophied; in time tricuspid incompetency follows. As a sequence of this we shall have a regurgitation through the right auriculo-ventricular opening and a consequent right auricular enlargement followed by general venous engorgement. As there will be a diminished blood supply to the left ventricle, few changes will be observed here, except a tendency to its gradual diminution in function and size. If it shows any dilatation or hypertrophy, the cause will be found in a coexisting mitral regurgitation. The increase of the cardiac weight will be slight, but the hyperemic and sclerotic changes in the pulmonary parenchyma will be pronouncel, producing cyanotic induration, thrombosis and hemorrhagic infarctions.

Symptoms.—There are no prominent symptoms when there is a *fair degree of compensation.* Any excitement or exertion will, however, cause *slight dyspnea* and *palpitation.* Whenever recurrent attacks of endocarditis occur the vegetations are liable to be detached and carried into the blood current, causing cerebral embolism, which will be manifested by symptoms of aphasia or hemiplegia. *Pressure* by the enlarged left auricle on the recurrent laryngeal nerve may cause paralysis of the left vocal cord. *Anemia* and left intercostal neuralgia are often noticed. When *compensation fails* the symptoms simulate those of mitral insufficiency. The pulmonary engorgement will now cause a depressing dyspnea in case of great exertion. Edema of the lungs may be so pronounced that a disturbing cough will be followed by hemoptysis. The dyspnea will then be constant and the pulse will be arrhythmic. Edema of the limbs and general anasarca are not extensive unless the tricuspid insufficiency is extreme. Should this condition exist we may find the same portal engorgement which follows mitral insufficiency. Intercurrent elevations of temperature will be symptomatic of the endocardial attacks.

Physical Signs.—*Inspection*—The apex beat will be displaced only in case of great enlargement of the right ventricle or associated hypertrophy of the left ventricle. In children the lower part of the sternum and the fifth and sixth left cartilages may bulge somewhat on account of the right ventricular hypertrophy. The *increased pulmonary tension* may create a visible pulsation in the second, third and fourth interspaces to the left of the sternum if the chest wall is thin. Sometimes it will be diffused along the right border of the sternum. *Epigastric throbbing* is commonly present. Following *compensatory failure,* the cardiac impulse will be undulating and feeble and the jugular veins will also pulsate. *Palpation* will reveal a *presystolic thrill* at the third and fourth, sometimes also at the fifth, interspaces within the nipple line. It is described as a purring fremitus which gradually increases in volume and intensity, and terminates abruptly with the apex impulse. This is pathognomonic of mitral stenosis and may be felt best when the patient assumes the left lateral decubitus. We may also feel the *cardiac throb* in the epigastric region. The *radial pulse* is small, compressible and irregular in proportion to the imperfect filling of the left ventricle and the diminishing power of the right ventricle. The sphygmographic tracing is extremely irregular.

Percussion.—The area of *cardiac dulness* is increased to the right and up along the left sternal margin to the second rib. There is no increase of dulness downward to the left on account of the inactive left ventricle. This occurs only when there is associated left ventricular hypertrophy as the result of a coexisting mitral insufficiency.

Auscultation.—The ear may detect a rough *presystolic murmur* in a limited area about the normal apex beat which has also been described as rumbling, vibratory, churning or purring in character. It *appears* and *disappears* in accordance with the strength of the auricular systole; as the right ventricle grows weaker it may become atypical or permanently disappear; if the strength of the left auricle and right ventricle is restored it will reappear. The *peculiar feature* of the murmur is noticed in its abrupt termination with the first sound in a short, sharp and snapping sound. The *aortic second sound* is weak, while the *pulmonary second sound* is markedly accentuated and distributed over a considerable area. The second sound may also be *reduplicated* and the murmurs of mitral and tricuspid insufficiency may be intermingled.

Diagnosis.—The features of this disease are defined by the *presystolic* thrill over a circumscribed area near the apex, the *increase* in precordial dulness upward and to the right, the *rough* and *purring* character of the murmur, as well as its *sharp termination* with the first sound and the *accentuated* second pulmonic sound. It will be easy to differentiate the symptoms of *mitral incompetency,* which presents a systolic murmur, soft and blowing in character, covering a greater area of transmission and synchronous with the pulse.

Prognosis.—The trend of all valvular diseases is toward an unfavorable termination and yet with proper physical and medical care it is possible to develop a safe chronic stage in which immediate death is not probable, except from some undue strain. If the protecting power of hypertrophic compensation is developed, there is slight danger of a serious result.

AORTIC INSUFFICIENCY.

The definition of this disease will refer to an imperfect approximation of the aortic segments, due to a diseased condition which causes contraction, shortening, curling or calcification of the valves. This permits a regurgitation of blood into the left ventricle. It has been designated in the past as "Corrigan's Disease." Aortic incompetence and aortic regurgitation are the other terms applied.

Etiology and Pathology.—This disease occurs most frequently among those in *middle life* who are engaged in active occupations—mental and physical. Prolonged and severe *muscular exertion,* too close and too long application to business, which favor the development of arteriosclerosis are important etiologic factors. *Syphilis,* gout, nephritis, blood poisoning, endocarditis, aortic aneurysm, alcoholism and lithemia are predisposing factors. *General arteriosclerosis,* affecting the coronary arteries, may favor myocardial degeneration and predispose to valvular impairment.

The *structural changes* are similar in character to those of mitral insufficiency. On account of arteriosclerosis we may find vegetations and calcareous deposits on the valve margins; the valves may be curled, or adherent to the aortic wall, or ruptured from some severe muscular effort, any of the above mentioned conditions preventing their perfect approximation. There may also be a dilatation of the aortic ring due to aortic aneurysm or to sclerosis of the aorta at its beginning, which prevents the normal valves from closing perfectly. The *mechanical results* of aortic regurgitation will be first manifested in dilatation of the left ventricle due to over filling with blood regurgitating from the aorta and that coming from the left auricle. The effort of the ventricle to expel this increased quantity of blood results in hypertrophy and dilatation. The *excessive hypertrophy* of the left ventricular walls in this disease gives rise to the term "cor bovinum."

The *primary effect* of aortic regurgitation is to decrease the amount of blood in the systemic arterial system and to increase that in the systemic venous and pulmonary circulations; yet because of the excessive hypertrophy of the left ventricle and its consequent increased power we may have increased arterial tension with danger of apoplexy and aneurysm. Later, as the coronary arteries fail to receive sufficient blood, a secondary dilatation occurs from fatty or fibroid degeneration.

Mitral insufficiency may accompany this disease or result from the secondary dilatation of the left ventricle. When it is present there will naturally follow the pathologic enlargement of the left auricle and right ventricle, evidenced by pulmonary and venous congestion.

Symptoms.—*Pronounced symptoms* will be unnoticed so long as a compensating hypertrophy protects the left ventricle. *During the process of hypertrophy* in case of any undue excitement of the heart, the patient may complain of a throbbing headache, vertigo and tinnitus aurium, together with a dull precordial pain which radiates into the neck and arms. *Slight dyspnea,* palpitation and anginal symptoms may follow intense excitement or exertion. *Angina pectoris* is often the cause of sudden death. *Flashes of light* before the eyes, redness and heat in the skin, with copious sweating, may follow the dilatation of the peripheral blood vessels. The *pulse* is small and jerky, owing to the imperfect filling of the arteries. The *systemic* and *pulmonic* veins will be congested unless proper hypertrophy of the left ventricle overcomes this.

Failure of compensation results in dyspnea and favors pulmonary and general venous stasis. *Cough* and occasional hemoptysis are sometimes prominent symptoms. *Dropsy,* appearing first in the feet, or *cerebral embolism* may follow. This is not so great as in mitral insufficiency. However, as relative mitral incompetence very frequently follows aortic regurgitation, we may observe the extreme symptoms of that disease. In that case the anasarca may become so troublesome that the patient will be able to breathe with comfort only when sitting up in bed. *Cardiac asthma* may add to the dyspnea. *Embolism* and congestion of various organs, together with febrile signs and profound prostration, may now occur. Marked restlessness and in time mental depression may terminate in suicidal impulses unless sudden death comes to the patient's relief.

Physical Signs.—*Inspection—Precordial bulging* is often observed in young patients and we may notice a wide and forcible area of cardiac impulse in the typical case. The *carotids,* temporals, brachials, radials and other visible arteries pulsate violently. *Capillary pulsations* may be detected and sometimes a venous pulse will be observed in the extremities. *Epigastric pulsation* will be present when there is excessive enlargement of the left ventricle or more particularly when a relative tricuspid insufficiency has led to hypertrophy of the right heart.

Palpation.—The *apex beat* is forcible and heaving, unless dilatation overcomes the hypertrophy, when it is weak and wavy. It may be felt in the sixth and seventh interspaces to the left, often as far as the mid-axillary line. A *diastolic thrill* is sometimes detected at the aortic interspace. The *peculiar pulse* known as the Corrigan or "water-hammer" pulse is defined as an abrupt and jerky pulse which suddenly recedes from the fingertip. It represents a sudden filling of the artery, followed by a rapid collapse of the vessel because the tension is not maintained. We shall observe this better when the arm is elevated above the head. A sphygmographic tracing will show an abrupt and excessive rise due to contraction of the hypertrophied ventricle, and this will be succeeded by a rapid fall as the blood regurgitates into the ventricle at the same time that it passes into the capillaries. This peculiar pulse wave will be modified as other lesions complicate the aortic insufficiency.

Percussion.—*Cardiac dulness* will extend downward and to the left in accordance with the left ventricular hypertrophy and dilatation. Later, because of the resulting mitral insufficiency left auricular dulness may extend upward on the left and to the right because of right heart hypertrophy and dilatation. Dilatation of the aorta gives dulness in the aortic area.

Auscultation.—The stethoscope detects a *diastolic murmur* with and following the second sound, heard generally in the aortic interspace to the right of the sternum. This sound may be transmitted downward toward the apex or the ensiform cartilage; it may also be heard in various parts of the chest and be reflected along the vessels of the neck; it is possible to locate it posteriorly to the left of the spinal column. In *quality* the murmur is soft, blowing and often musical; sometimes it is rough and loud; again it may be almost inaudible. There is sometimes an accompanying systolic murmur that produces the so-called "double aortic" sound, which is transmitted into the cervical region. More frequently, however, we shall find accompanying this a soft systolic apex sound due to a secondary mitral insufficiency. A *presystolic mitral* murmur over a limited area has been described by Flint. This occurs rarely as a result of extreme dilatation of the left ventricle. *Double murmurs* may be discovered by auscultation over some of the peripheral arteries. Duroziez was first to notice this in the femoral artery and it is now considered by some authors as a pathognomonic sign of aortic regurgitation. Over the carotid and subclavian arteries a systolic murmur often displaces the first sound; and if the regurgitation is free the normal second sound is absent.

Diagnosis.—The *physical findings* must be studied in connection with the condition of the vascular system. The diastolic murmur heard over the aortic interspace, the throbbing arteries, the peculiar pulse and the marked hypertrophy of the left ventricle are reliable signs on which to make a diagnosis when they are found in a patient past middle life. If the predisposing causes—alcoholism, syphilis, gout and other debilitating diseases—are also found, they will help to confirm the above diagnostic points. The consequences of unusual or sudden *physical strain,* if associated with these typical symptoms, will add to the confirmation of this diagnosis. The *relative mitral insufficiency* should always be taken into consideration in cases of long standing, particularly when the left ventricle is excessively dilated. So, too, we must differentiate in cases of aneurysm of the arch of the aorta.

Prognosis.—The prognosis will depend upon the predisposing causes, the general health of the patient and systematic treatment. Much depends upon the character and degree of the compensating hypertrophy. When *endocarditis* is the contributing factor we may expect a reasonable amount of relief if compensation follows the early use of remedies. Should signs of myocardial degeneration appear, we can not expect much improvement. General *arteriosclerosis* should always suggest an unfavorable prognosis. Should stenosis develop a certain amount of amelioration may ensue provided no myocarditis exists, but this is a rare possibility. Death is liable to come suddenly as a result of paralysis of the left ventricle during diastole. A fatal termination is more liable to take place from gradual cardiac asthenia or from acute pulmonary edema.

AORTIC STENOSIS.

This is defined as a narrowing of the aortic orifice due to inflammatory changes which affect the valve structure or the ring itself. It is a rare instance when it exists as a congenital defect, and is not commonly an independent lesion. Valvular incompetency is generally an associate or subsequent development.

Etiology.—Males are said to be subject to this disease oftener than females. The young may be afflicted, but it is more liable to occur during advanced life when the atheromatous process is in evidence. The etiologic conditions incident to endocarditis and the degenerative changes which lead up to the stenosis have been completely described in the chapters on that subject.

Pathology.—*Structural occlusion* may follow the adherence of the aortic cusps. This is an inflammatory condition, though it is a congenital possibility. Because of rigidity the valves do not open properly and thus form an obstruction to the normal blood current through the aortic opening. Again, vegetations may appear on the ventricular surface of the segments and interfere with the closure of the valves. As a *gradual sclerosis* or calcification naturally follows, the occlusion becomes more pronounced. *Vegetations* may develop in the sinuses of Valsalva. In a majority of cases with the foregoing pathologic conditions, aortic incompetency will be discovered, thus showing that stenosis is not an independent entity. In some instances the aortic ring itself will be the seat of accretions which obstruct the normal blood flow; and the same pathologic changes may extend for some distance up the ascending aorta also and favor this.

The *mechanical results* of this perverted anatomy of the heart will be shown in left ventricular hypertrophy. This is caused by the unusual muscular activity necessary to pump blood through the narrowed aortic orifice. The hypertrophy develops slowly and eventually gives way to extreme dilatation and a relative mitral insufficiency. Finally the general cardiac hypertrophy and the passive congestions which follow the other cardiac lesions will be recognized. The coronary vessels may be deprived of their normal blood supply by a gradual sclerosis until myocardial degeneration ensues.

Symptoms.—The *clinical features* will depend very much upon the degree of stenosis as well as upon the extent of compensating hypertrophy. If the pathologic changes are slow in development, there may be no decided symptoms. As the left ventricle fails in its effort to propel blood into the arterial tree, a certain amount of dizziness, headache and pallor will be noticed as evidence of cerebral or peripheral anemia. *Attacks of syncope* and epileptiform seizures are not uncommon at this period. As *aortic incompetency* is generally associated with stenosis, the symptoms of this lesion will appear. In case also of mitral incompetency we shall have the supplemental symptoms of that disease with venous stasis, left ventricle weakness and irregular pulse. *Edema* then will be manifest in the feet and gradually extend upward, until anasarca is quite general. By dislodgement of fibrinous clots, embolism in the brain, spleen, kidneys or other organs may occur.

Physical Signs.—*Inspection.*—*Pallor* will be an early symptom depending on diminished general circulation. *Cyanosis* appears only as there is failure of left ventricle compensation. The *apex beat* is displaced downward and outward in proportion to the enlargement of the left ventricle. The beat is usually slow, forceful and heaving, though less frequently it may lack in strength. If the chest wall is rigid of the lungs are emphysematous, the cardiac impulse may be indistinct. Precordial bulging may be detected when the chest walls are thin and the cardiac enlargement is pronounced.

Palpation.—This will disclose the *forcible* and *heaving* precordial impulse which is explained by the powerful contraction of the left ventricle necessary to overcome the aortic obstruction. As this impulse becomes weaker, it will be an evidence of failing compensation of the left ventricle. A *systolic thrill* will be felt in the aortic interspace and at various points over and above the cardia. The *pulse* is weak and small, its size denoting invariably the degree of stenosis. Aortic and mitral incompetency will modify the character of this pulse. The sphygmographic tracing shows an oblique line of ascent, a rounded summit, a gradual descent and the secondary waves indistinct.

Percussion.—The *area of dulness* is increased toward the left and downward, commensurate with the degree of left ventricular hypertrophy. Dulness to the right may be detected whenever the mitral lesion favors enlargement in this direction.

Auscultation.—A *systolic murmur* of harsh quality may be heard in the aortic interspace. As this represents an obstruction which interferes with the normal sound made by closure of the aortic segments, it will be heard at the end of the first sound or just preceding the second sound. The direction of this murmur will be along the large blood vessels. When *compensation fails* this sound will lose much of its force and harshness. The second sound is faint and often inaudible, on account of the diminished blood tension in the aorta. An *aortic incompetency* will give an additional diastolic or double murmur. A soft apical systolic murmur may appear after relative mitral insufficiency ensues.

Diagnosis.—A *systolic thrill* at the base, a small tense and characteristically slow pulse, with signs of left ventricular hypertrophy, and a rough loud systolic murmur at the aortic interspace, should make the diagnosis clear. The *musical quality* is another feature which is quite distinctive. The *differential diagnosis* must exclude (1) sclerosis of the aorta or its valves without obstruction; (2) aortic aneurysm; (3) accidental or anemic murmurs. In *aortic sclerosis* we shall find the hard peripheral arteries, accentuation of the aortic second sound, general cardiac enlargement and frequently a history of excesses or syphilis. In *aortic aneurysm* we may obtain a history of some severe strain, followed by pain, dyspnea and cough, inequality of the carotid and radial pulse, cardiac displacement, expansile pulsation, with bulging in the aortic area, and the characteristic bruit. The X-ray may aid the differentiation when there is much doubt. *Anemic murmurs* will be diagnosed by the absence of rheumatic history, the very slight hypertrophy of the left ventricle, the unchanged aortic second sound, the softer quality of the murmur, the greater frequency of the pulse, the presence of other accidental murmurs and the manifest anemia. We shall also find the majority of these cases among women.

Prognosis.—The prognosis in this disease is similar to that in all of the cardiac valve lesions. When the compensation develops favorably the danger of immediate death is not great. If proper caution against overexertion is observed and particularly if no infectious disease develops we may safeguard the heart's action by appropriate treatment. The etiology is an important factor in this respect. Anything which favors a degenerative process conduces to an unfavorable condition. When compensation *begins to fail* there will be steady progress toward a fatal termination. Death does not, however, usually come suddenly; it results rather from the progressive cardiac asthenia as observed also in the mitral lesions.

TRICUSPID INSUFFICIENCY.

The insufficiency of the tricuspid valves is known and spoken of also as tricuspid incompetency and tricuspid regurgitation, all of which expressions are illustrative of the anatomic perversion which affects these valves and prevents the normal flow of blood through the right auriculo-ventricular opening. It exists occasionally as a structural insufficiency, but more frequently it is a relative insufficiency. It is also the most common lesion of the right heart.

Etiology.—*Structural defects* are most frequently the result of right heart endocarditis occurring during fetal life or in early childhood. In the latter case the same etiologic factors which pertain in the other valvular diseases will be recognized. The *relative form* is the prevailing one and arises as a result of high tension in the pulmonary system due to interstitial pneumonia, chronic bronchitis, pulmonary tuberculosis, emphysema and possibly to other diseases. This is particularly true of the obstructive and regurgitant diseases of the left heart. The *insufficiency* is bound to follow when the right ventricle is no longer able to endure this back pressure and yields to dilatation. *Renal* or *hepatic cirrhosis,* or any disease which augments the blood tension in the pulmonic circulation, will finally throw such pressure upon the right ventricle that it—particularly when there is mural degeneration—must eventually dilate until the ring stretches sufficiently to permit a regurgitation. Severe *muscular exertion* and toxic agents may be the occasional means of direct affection of the right ventricle when the nutritive quality of the blood is lowered by anemia or similar disorders.

Pathology.—We shall first discover the pathologic conditions of the left heart and the lungs which lead to this tricuspid regurgitation. The *right ventricle* is dilated, though evidence of muscular hypertrophy may be found. The *valve segments* are elongated on account of the prolonged pressure; the *papillary muscles* are inelastic and the right auriculo-ventricular opening will be excessively dilated. This dilatation, as the result of constant back pressure, may extend to the veins which enter the right auricle. The *myocardium* will show evidences of prolonged stasis and degeneration. For these pathologic reasons we may regard the tricuspid regurgitation as a "safety-valve action" which, to a great extent, spares the right ventricle. The right auricle is dilated and its muscular walls may be compensated by some muscular hypertrophy.

The *mechanical results* may be accounted for as follows: When the tricuspid leakage permits an undue reflux of blood into the right auricle, venous stasis will be a natural result. There will also be a lessened blood supply to the pulmonary arteries and this may lead to general anemic symptoms. The condition of the pulmonary circulation will be such that the right ventricle must at first dilate and be compensated by gradual hypertrophy. Inasmuch, however, as there is no large chamber to give compensating protection to the right ventricle, its walls gradually become thin until the ventricle assumes enormous dimensions in some cases. The dilatation of the right auricle is the only compensating relief which may be obtained in this disease, aside from ventricular hypertrophy.

Symptoms.—*Engorgement* of the systemic veins will constitute an early symptom. *Cyanosis* and turgescence of the superficial veins will show this. *Passive engorgement* of the abdominal viscera, particularly the liver, usually follows and edema starting in the feet soon terminates in general anasarca. This does not always exist in every case. Hydrothorax is a natural sequela in this disease. *Exhaustion,* dyspnea and cough are common symptoms in the later stages. Other symptoms will pertain to the lesions of the left heart which are so frequently the primary cause.

Physical Signs.—*Inspection.*—The most characteristic sign is the *pulsation of the jugular veins* during each cardiac systole, more marked on the right than the left side. In rare cases the subclavians may pulsate. This is due to the backward flow of blood into the right auricle at each ventricular systole. *Palpation.*—Pressure on the vein will confirm this if the return flow from the right ventricle is detected by the finger after the blood has been pressed out of the vein. The vein will fill by "jets synchronous with the heart-beat." This pulsation may possibly be communicated by the throbbing of an adjacent artery; if this be true light pressure upon the vein will not stop the pulsation. We may still further test this by pushing the artery away from the vein. The *location* of the apex beat will vary in accordance with the primary heart lesion. Besides the jugular throbbing *epigastric heaving* will be felt by the hand; this is caused by the increased action of the enlarged right ventricle. A *venous pulsation* also extends to the liver and may be felt by placing one hand over the anterior hepatic surface and the other pressing up from behind; it will be recognized as an expansile pulsation. The *radial pulse* varies in its character in accordance with the pathologic condition; it may be small and weak or irregular and accelerated from time to time. Popoff claimed to have detected a diminution in the size of the right radial as compared with the left. This was supposed to come from the pressure of the dilated right auricle upon the right subclavian artery.

Percussion.—*Dulness* will be increased to the right and upward, often as high as the second interspace; it may also extend downward into the epigastric region. This will vary in extent according to the complicating lesion.

Auscultation.—A *systolic murmur,* heard best over the base of the ensiform cartilage, is a typical diagnostic sign of tricuspid insufficiency. This, however, is subject to variation. It may be displaced to the right of the ensiform cartilage and upward or to the left, dependent upon the degree of hypertrophy and dilatation. In character the sound is soft and short and is modified by bruits of other cardiac lesions. The *pulmonary second* sound is naturally enfeebled, though at times it may be accentuated.

Diagnosis.—The *venous pulsation* in the jugular and other areas is the most suggestive sign for diagnosis. The confirmation of *hepatic pulsation* by bimanual palpation is extremely valuable. If the murmur can be *definitely located* in the tricuspid area, our opinion will be more positive. If, however, this murmur is indistinct, it may be only a secondary condition following mitral insufficiency.

Prognosis.—Whenever tricuspid insufficiency is relative to mitral or other lesions the outcome will depend upon the causative factors. Unless the right ventricle is unable to sustain itself by muscular hypertrophy, we should not look for sudden death from this lesion alone. Generally speaking we should regard inflammatory invasion of the right ventricle with great concern inasmuch as the venous stasis is liable to produce serious results in remote parts.

TRICUSPID STENOSIS.

Tricuspid stenosis is such a rare disease that it seems unnecessary to enter into a long discussion of its principal features. It will be noticed more frequently in females, and during the early periods of life. Rheumatic fever is no doubt a prominent factor in its development. By some the existence of tricuspid stenosis is doubted, though recent autopsies have confirmed its occurrence. It exists usually as a secondary condition, though it is a congenital possibility. Organic diseases of the left heart most frequently predispose to, and mitral stenosis is most often associated with it. Vegetations and ring accretions are found here as in the left heart when endocarditis is a cause.

The *pathologic changes* are dependent both upon the tricuspid stenosis and the primary causes. When the right auriculo-ventricular opening is obstructed the chief anatomic abnormality will be found in the right auricle. As this chamber is unable to empty itself dilatation and hypertrophy follow in natural order. Inasmuch as the right auricular walls are thin, very little compensatory hypertrophy can take place and therefore dilatation is both extreme and dangerous. The right ventricle, receiving a smaller amount of blood, is diminished in size. When there is a conjoined mitral defect there will be a combined hypertrophy and dilatation of the right ventricle.

The *symptoms* are so occluded by the combined affections that a positive diagnosis is rarely made except at the autopsy. *Stasis* in the systemic veins will be evidenced, but this differs in no respect from the similar symptoms of mitral disease. As the blood supply to the lungs is cut off, *pulmonary congestion* and *dyspnea* are not pronounced after exertion. *Jugular distention,* hepatic congestion, cyanosis, edema and ascites must naturally result from the surplus of blood in the right auricle.

The *physical signs* are not very significant. *Inspection.* Aside from the feeble, visible jugular impulse and the general appearance of venous stasis, we shall observe but little of diagnostic value. *Palpation* may reveal a presystolic thrill in the neighborhood of the ensiform cartilage, and the pulse will be small, weak and irregular. *Percussion* will distinguish an increased dulness over the right auricle, but this is not a positive proof of primary tricuspid stenosis. *Auscultation* may present a presystolic murmur, heard best over the lower part of the sternum and up the right border. Unfortunately there is no positive proof that this is generated from the tricuspid orifice. It will be inferred from the foregoing statements that an absolute *diagnosis* is not a possibility. We may say with equal truth that the *prognosis* is clouded by the same uncertainty.

PULMONARY VALVE INSUFFICIENCY.

This is also a disease of unusual rarity. It has been attributed to congenital malformation and to acute and chronic endocarditis. Adherence of the segments and the sclerotic process are the pathologic factors, the same as in other cardiac lesions. Many of the murmurs are secondary to blood changes and are purely functional. Atheroma of the artery is often a pathologic cause. The result of the lesion will be observed by the hypertrophy and dilatation of the right ventricle. A relative insufficiency of the tricuspid valve may follow.

The *physical signs* are not very reliable, yet a *diastolic murmur* may be heard over the pulmonic interspace. As this is transmitted downward and to the right it may suggest aortic regurgitation, yet the peculiar pulse and other diagnostic signs of this lesion are not present. *Pulmonary insufficiency* will invariably be followed by right ventricle dilatation and hypertrophy, while *aortic regurgitation* will not create right ventricle enlargement unless there is an accompanying mitral insufficiency. As the right ventricle is finally unable to resist this undue tension, the tricuspid approximation will be disturbed and tricuspid insufficiency will probably develop. The ensuing venous stasis, together with other ill-defined symptoms, will tend to obscure the real lesion, while our attention is given to the complicating cardiac disease.

PULMONARY VALVE STENOSIS.

This is also a disease of extreme rarity. It is due most frequently to congenital malformations. Atheroma, myocarditis, ulcerative endocarditis and direct violence are claimed as causes. Hypertrophy of the right ventricle and the succeeding dilatation, with relative tricuspid insufficiency present symptoms of cyanosis and venous distention. *Physical examination* will reveal a systolic thrill heard best over the pulmonic interspace. This sound will be harsh, superficial and limited to a small area, though it may extend upward towards the left shoulder. The pulmonic second sound is weak. This lesion predisposes decidedly to pulmonary tuberculosis.

COMBINED VALVULAR LESIONS.

The foregoing descriptions pertaining to single heart-lesions and their associate compensating conditions, are characteristic of the diseases which affect the valves and the cardiac orifices. We shall find, however, that these lesions do not always occur alone. Aortic regurgitation and obstruction, or possibly mitral regurgitation and obstruction coexist much more frequently than is recognized. This means that the endocardial inflammation may act as a pathologic factor to create sclerosis of the valve segments and their eventual shrinkage and at the same time produce accretions which narrow the orifice. We shall also find the lesion of one orifice combined with that at another orifice. All of these perversions exist as a result of the morbid anatomic change, yet they act to a great extent as compensatory factors to maintain the cardiac function.

MITRAL STENOSIS AND MITRAL INSUFFICIENCY.

In this we shall observe the presence of both a constricting obstruction and the insufficiency from non-approximation, due alike to the same pathologic cause. The mechanical effect upon the heart differs somewhat from the results of a single lesion. The left ventricle is not dilated so much as in simple insufficiency, nor is it decreased in size as occurs from the inactivity of this part in stenosis alone. The left auricle is not so liable to extreme hypertrophy as in stenosis, nor is it so dilated as when regurgitation is freely established. The two lesions together tend to overcome the pronounced changes of each.

The *physical findings* combine and limit the symptoms of each lesion. The apex beat is less forcible and not displaced so much as in mitral regurgitation, while the stenotic thrill is less intense. Dulness is increased more to the right and the pulmonic second sound shows a marked accentuation. Auscultation shows both a presystolic and a systolic murmur, the latter being slightly prolonged, while the former is often short and sharp.

MITRAL STENOSIS AND AORTIC STENOSIS.

In the combined stenosis of these two orifices we find that the left ventricle is small on account of the decreased amount of blood received from the left auricle. At the same time its muscular wall is thickened by its unusual effort to propel blood through the narrowed aortic ring. The walls of the left auricle and right ventricle are greatly hypertrophied and dilated because the blood is retained in these chambers. Pulmonary stasis in this case is followed by an extensive general venous stasis. Cyanosis and dyspnea are, therefore, characteristic symptoms.

The *pulse* is generally small, weak and slow, depending in character upon the amount of hypertrophic compensation. The apex beat is not strong, unless it is made so by excessive right-heart hypertrophy. A presystolic thrill is detected by palpation and this is confirmed by auscultation at the apex. A similar harsh sound should be detected at the aortic interspace slightly in advance of the second sound. The pulmonary second sound is also accentuated. The disease is exceedingly serious and the prognosis is always unfavorable, inasmuch as compensation is rarely maintained.

MITRAL STENOSIS AND AORTIC REGURGITATION.

The cardiac findings in this disease will vary with the degree of the underlying pathology. If they are similar in degree, the secondary effects are as follows: The left auricle and right ventricle are hypertrophied and dilated on account of the mitral stenosis. The left ventricle shows a similar tendency, though it will be greatly modified by the limited amount of blood entering this chamber through the narrowed auriculo-ventricular opening. A careful study of the predominating lesion will be necessary to establish the physical signs in each case.

Inspection shows an apex beat displaced to the left and downward, but less than in typical aortic insufficiency. Epigastric and superficial venous pulsations are present in proportion to the mitral stenosis. *Palpation* reveals a prolonged presystolic thrill and an apex beat somewhat less forcible than in normal aortic regurgitation. The pulse is small and weak on account of the stenosis and the artery collapses as in the aortic lesion. *Percussion* discloses increased cardiac dulness in all directions. The left auricle and the right heart are enlarged because of the stenosis, and the left ventricle is slightly enlarged as a result of the aortic regurgitation. *Auscultation* discloses a presystolic murmur at the apex and an accentuated pulmonic second sound due to the stenosis. In combination with this we hear a diastolic bruit in the aortic interspace transmitted downward and to the left, with a feeble second sound. The femoral artery should be auscultated for confirmation by the sharp systolic sound or the double murmur heard over this vessel.

AORTIC REGURGITATION AND MITRAL REGURGITATION.

These two lesions are observed quite frequently in combination. They appear rarely as the result of structural alteration, but they are often observed when the mitral insufficiency follows aortic insufficiency of long standing. The mitral lesion is then called a relative incompetence of the left auriculo-ventricular valve. Formerly this disease was considered a grave one, but recent experience seems to confirm the opinion that it will respond favorably to early treatment. If the heart maintains the essential compensatory hypertrophy the two ventricular leaks may be held in subjection. If the muscular tone is lost or if any myocardial degeneration complicates, the prognosis is very unfavorable. It is also evident that the heart, with this double regurgitation affecting the left ventricle, will stand much less strain than with a single lesion. Sudden cardiac failure is, therefore, always to be feared in these cases.

The *symptoms* modify those of each single lesion. The mitral regurgitation diminishes the force of the aortic regurgitation since some of the blood flows back into the left auricle. The arterial wave is lessened, and as a result we shall notice a tendency to arterial anemia. Venous congestion will depend upon the hypertrophy and dilatation of the left auricle and right ventricle. Ordinarily the disturbance of the venous system is marked and the heart attains a large size. The pulse is small and collapsible. Dulness is increased, both to the right and the left. A diastolic murmur is heard at the aortic interspace and a systolic murmur at the apex. If the aortic regurgitation is of long standing, it may be difficult to detect a mitral regurgitation. The general appearance of the patient shows the effects of passive congestion.

AORTIC STENOSIS AND AORTIC REGURGITATION.

Aortic regurgitation is quite likely to follow aortic stenosis. The vegetations and accretions which dispose to stenosis and the consequent obstruction at the ring are generally attended by some thickening and rigidity of the valve segments. This interferes with complete closure and allows a regurgitation into the left ventricle. One of the cusps is also liable to be perforated when the others are intact which likewise predisposes to regurgitation. It is necessary in making this diagnosis not to be deceived by the roughness of the aortic regurgitant murmur, which often simulates the stenotic sound. The outcome in this combination of lesions is anything but encouraging. If the regurgitation exceeds the stenosis, the left ventricle may compensate sufficiently to save the heart for some time. If stenosis is more pronounced, the lack of ventricular hypertrophy will tend to make even the slightest dilatation dangerous.

The *physical signs* vary in accordance with the degree of each defect. The cardiac impulse is not so forcible or extensive as in simple regurgitation, and the apex beat simulates that observed in stenosis. Compensating hypertrophy is not unusually marked. The pulse is slow and small, but not so characteristic in this respect as in simple stenosis; it is collapsible, but not so much so as in aortic regurgitation alone. Capillary pulsation and the double femoral souffle are not present. Two murmurs will be discovered,—an intense and rasping systolic and a milder diastolic sound. As the disease progresses, the heart is liable to other degenerative changes which will modify these physical findings.

THE TREATMENT OF VALVULAR DISEASES.

The treatment of valvular diseases of the heart necessitates a thorough study of each case, the predisposing causes, the peculiarities of the patient and the individual features of the lesion. This will be undertaken under three distinct heads: (1) prophylaxis, (2) during the stage of compensation, (3) during the stage of non-compensation.

Prophylaxis.—It is a common mistake on the part of both patient and physician to think of treatment only when pathologic signs are in evidence. In reality the first thought about the heart should go back to the infectious diseases or the cardiac strain, which predispose. Too many cases of rheumatic fever, pneumonia, influenza, etc., are treated with no thought of, or at least with no caution to the patient about, a possible cardiac lesion in the future. In every infection the physician should watch carefully lest the heart suffer from this cause. In fact, this is the time when the treatment of the heart should be first instituted. It is at this time that rest, diet and medicinal measures will have the most telling effect. If we give sufficient attention to heredity, we may obviate the tendency to heart weakness which prevails in certain families. If we look more carefully into the habits of our patients, we may correct certain errors which eventually lead to this predisposition. Too many people indulge in the excessive use of tobacco and alcohol without the advice of their physician regarding its ultimate danger to the heart. Too many ignore the consequences of physical excess and strain until the dangerous signs of cardiac failure appear. Thus it is evident that our best work must be done when the heart disease is in its incipiency.

During Compensation.—It is during this period of valvular disease that the most effective treatment is possible. At this time nature is doing her best to protect the heart, and our assistance may preserve its contractile power. Every influence which unduly excites the normal function or aggravates the lesion must be removed. Exercises and occupations which tend to give a healthy stimulation may be judiciously tolerated, but any strain which accompanies violent exercise must be interdicted. When a lesion throws any functional stress upon any particular part of the heart muscle, a compensating hypertrophy develops for the sake of its protection. If we aid this physiologic effort, the heart may continue in this stage a long time. Much depends upon the character of the irritating cause. If the rheumatic, gouty, syphilitic or alcoholic taint exist, we may correct these by our general treatment for such causes. When myocardial degeneration is a factor, our treatment must be more painstaking, and greater protection against muscular tension must be instituted. The secondary effects upon the circulation must always be kept in mind. Stenosis of the mitral and aortic openings will require more care than the regurgitations at the same points. The degree of incompetence and the extent of the valvular lesion will modify the treatment. A slight leakage may be naturally compensated, but an excessive regurgitation will demand much help and care. Fatigue, emotional excitement, fright and the use of stimulants will cause the greatest harm at this period. Healthful exercise and vocations which do not overstimulate the heart may be permitted. Systematic rest and relaxation from business cares must be at times authoritatively insisted upon.

Diet is a matter of essential consideration. A moderate amount of food—usually proteid—should be taken at regular intervals. The stomach should never be overloaded. Carbohydrates, which tend to form gases, should be cautiously allowed. Much, of course, depends upon the patient's occupation, idiosyncrasies and the digestive function. As anemia is so frequently a complication, it will be necessary to supply the best possible nutrition. Rest from arduous duties, often in bed, is essential for the digestive function as well as for heart tranquillity. The healthy action of the liver, and other adjuvant organs of digestion, are as important as the normal movements of the bowels. Neither should we neglect to give careful attention to all complicating diseases. When the patient is taught to be cautious in every habit we shall make the greatest gain in treatment. It will be necessary for this purpose to overcome many habits which relate to overeating, stimulants and tobacco, sexual indulgence, etc.

Warmth of the body must be constantly maintained. Measures for this purpose, of course, will depend much upon the physical peculiarities of patients. To some, woolen undergarments are a necessity, but others are made comfortable by lighter underwear and a proportionate increase of outer wraps. Occupation will decide this in many respects. Those residing in a cold climate may need a change to one warmer and more equable. High altitude must be cautiously considered in individual cases. Oxygen is an invaluable remedy, and we should never shut our patients up in a hot and poorly ventilated room. The cool sponge bath will, in the majority of cases, stimulate the circulation and induce warmth. Baths, of course, are vital for the sake of cleanliness, but the depression following a prolonged hot bath must be avoided. Turkish, Russian and even the Nauheim baths should not be employed at this stage of the disease.

Internal medication, and particularly stimulating drugs, are not usually essential at this period. Compensation is a physiologic change, and if we follow the instructions just mentioned, this will be accomplished with the aid of but little medication. No greater mistake can be made than to resort to the use of such remedies as digitalis at this time. Advice which embraces the foregoing suggestions and a placebo are often sufficient therapeutic measures. When we have removed the predisposing causes, our medication should apply to the anemic condition of the heart muscle and other pathologic findings. The treatment therefore should be, to a great extent, in accordance with the totality of symptoms. Individualization in each case is a necessity. Stimulation, if required, should be obtained only by the minutest doses of such remedies as *digitalis, nitroglycerin, strychnia, strophanthus* and other heart tonics.

Arsenic will naturally appeal to us as the best cardiac tonic we have for this stage. It is suggested by the general exhaustion, the restlessness, the dry skin and emaciation, as well as by the palpitation and cardiac irregularity. In our study of this remedy we should compare *aconite, spigelia, veratrum viride, kalmia, amyl nitrite, lilium tigrinum, phos. acid* and *collinsonia.* The tissue remedies will be of great value. *Lachesis, apis, crotalus, naja* and *rhus tox* must always be kept in mind. *Ferrum phos., chininum ars.,* and *china* will often be indicated. *Lycopus, fucus vesiculosus* and small doses of *cactus grand,* will invariably control the cardiac excitement. We must never overlook the beneficial results obtained by *valerianate of ammonia* in the second decimal potency. *Kali bromatum* may be similarly considered. When specific causes are at fault, we may properly employ *aurum, mercurius* and the *iodids.* The strength of these remedies will be settled by the demands of the case and the individual peculiarities. The third decimal potencies are generally sufficient, except in the use of *lachesis, naja, crotalus,* etc., when a higher potency may be employed.

During the Stage of Non-Compensation.—We have found in the ordinary case that the heart is able, by a gradual compensatory hypertrophy, to bear the strain of increased work put upon it. During this period only slight help is demanded of the physician. When, however, the exhausted muscle is unable to maintain its function, it will be necessary to resort to more active measures. When compensation fails, dyspnea and arrhythmia are in evidence, but inasmuch as the failure is gradual, these symptoms are not always pronounced. Thus the degree of compensatory failure varies in different cases, but when it becomes marked and constant we may know that vigorous treatment is necessary. In extreme instances the increased dyspnea and arrhythmia are accompanied by cyanosis, an irregular, small and compressible pulse and other signs of cardiac enfeeblement. In addition to these symptoms dropsy will gradually or rapidly appear. Patients in this stage will be unable to endure much exertion and the labored respiration will require absolute quietude.

Rest, therefore, is the first mandatory expedient of the treatment. This should invariably be obtained in bed with healthful and quiet surroundings. Mental rest is as essential as physical rest. Thus all business and domestic cares must be held in abeyance. If we keep the patient warm and quiet, we shall gain the first important requirement. It is a mistake also to relax this precaution until compensation is well established. This we shall recognize by the improved character and rhythm of the pulse, the disappearance of venous stasis, the increase in the amount of urine and a marked disappearance of dyspnea. In nearly all the lesions, but particularly in mitral disease, rest aids the forcible contraction of the left ventricle, diminishes the blood supply to this chamber and generally conserves the heart power. More than this, it relieves the overstrain upon the left auricle, the pulmonary vessels and the right ventricle. During rest the blood flows much more slowly and this gives the disabled left ventricle less work to do. This lessening of the number of contractions lengthens the diastole or period of repose, and at the same time the heart is working more quietly.

The *amount of exercise* which may be tolerated is a difficult matter to decide. In general we may permit its gradual resumption, though much depends upon the degree of compensatory failure. When there is complete failure, the patient should not be allowed to leave the bed. The resistance exercises are of modern introduction and no doubt give good results, but they should be administered only by an experienced attendant. It is a safe rule to follow that all exercises of this kind should be discontinued when any sign of dyspnea is observed. Oertel's plan of permitting patients to slowly climb a hill may be generally discouraged, inasmuch as the majority are liable to go to extremes in this respect.

The Nauheim baths have become very popular in the recent treatment of cardiac diseases, especially among the laity. That they constitute a favorable form of treatment we can not deny. These waters are impregnated with various chlorine salts,—particularly chlorid of sodium and calcium. The springs are also highly charged with carbonic acid which adds a stimulating effect, especially when the effervescing stream is allowed to flow upon the body. The chlorine salts are used in a limited percentage at first and no carbonic acid is used until the patient is accustomed to the bath. Rest is also an intermittent feature which is rigidly observed. Inasmuch as these baths are so far from American patients, only a few are able to avail themselves of such a privilege. The artificial baths, of a similar character, are now being employed satisfactorily. The general effect of a bath is that of a sedative to a weak heart, but its depressing reaction must always be watched with care. When cardiac inadequacy is marked, in extreme arteriosclerosis and where there is considerable edema, any bath other than a sponge is contraindicated.

The *diet* in the stage of failing compensation must be in accordance with the ability of the stomach and intestines to care for food. Much cardiac irritation is occasioned by gastric disturbance, and we should be sure that the stomach digestive tract can care for carbohydrates before we allow them in any quantity. The renal function must also be considered, for a disturbance here is a common complication in cardiac diseases. A non-nitrogenous and liquid diet, therefore, sometimes becomes a necessity. In our treatment of the heart we must remember that it is often necessary to direct our remedies to the gastric and intestinal conditions. In line with digestive disturbances we must be on guard constantly against anemia, which is so frequently an attendant upon valvular lesions. A vigorous diet may here be best and the usual remedies, iron, arsenic and iron-containing foods, should be employed in addition to other required treatment.

The *medicinal agents* which must be relied upon when compensation is failing or has failed, naturally come under the head of cardiac stimulants. There is no alternative at this time, yet we should be on constant guard lest we overdo in this respect. For this purpose *digitalis* is still a standard remedy, the infusion being used more particularly when cardiac dropsy is pronounced. Five to fifteen drops of the tincture, or one to three drams of the infusion, may be used as often as every three hours. As digitalis so frequently causes nausea, a form known as the "fat free" is a better preparation. Five to fifteen drops of this may be given from two to four times daily. Generally, after this remedy has been employed for a few days or weeks, we shall notice improvement. We must not stop here, for the remedy should be continued,—unless there are unfavorable reactions,—until there are signs of complete compensation. The cumulative action of this drug has often been overestimated, though it is a possibility. When there is a slowing of the heart's contractions to 60 or less per minute, when there is persistent nausea following its use, or when there is a sudden decrease in the amount of urine, then we may know that the toxic effect of digitalis is being manifested.

Another extreme effect of digitalis is to raise the arterial tension and this may increase the tendency to palpitation, and in case of arteriosclerosis injurious results may follow its protracted use. To overcome this it is well to use *nitroglycerin* (grs. 1/100) every two or three hours, should we find it necessary to continue the digitalis. To obviate this necessity it has become my custom of late to rely upon a compound tablet containing *digitalin* (gr. 1/100), *nitroglycerin* (gr. 1/100) and *strychnia* (gr. 1/50). This I administer as often as every three hours, and in extreme cases it acts best hypodermatically. I have saved many hearts from compensatory failure by this measure. In all cases with pronounced signs of cardiac hypertrophy it is best to rely entirely upon the hypodermatic use of this compound, as the internal administration does not give sufficient stimulation. *Strychnia* in this case gives an additional support to the heart by means of its action upon the cardiac motor ganglia, while the nitroglycerin compensates the increased arterial tension, which may be caused by the action of digitalis. After the above procedure has been continued until there is evidence of a safe compensation, it may be well to supplant this treatment by some indicated remedy which shall be a heart tonic. The compound tablet of *iron* (gr. 1), *arsenic* (gr. 1/100) and *strychnia* (gr. 1/60) four times daily, is a valuable prescription for this purpose. *Strychnia* as a heart tonic I do not consider alone sufficient in the stage of failing compensation. It may also be of service after *digitalis* has done its work.

Strophanthus is naturally comparable to *digitalis,* as it has a similar action upon the heart muscle. Its control of cardiac irregularity is often better than that of digitalis, though in some cases it is well to combine the two. In cardiovascular sclerosis or interstitial nephritis, this is a most valuable remedy. It is always useful when there is primary or relative weakness of the aortic valves. The dose is from five to ten minims of the tincture three or four times daily. This remedy, fortunately, has not the cumulative power of *digitalis* and hence it may be continued for a longer time without any gastrointestinal disturbance, though some patients are easily disturbed by it. *Caffein* is a remedy which has some. advocates. Dujardin Beaumetz claims its greatest service is during the last stage of cardiac disease, when we have exhausted the effect of other tonics. Hypodermatic use is sometimes made of a solution of *caffein* (gr. 30), *sodium benzoate* (gr. 35) and *distilled water* (m. 200). An injection of 15 to 20 minims of this is used at stated periods. *Spartein sulphate,* in grain doses of the first decimal trituration, every two or three hours, has given beneficial results when other remedies have failed. Quarter grain doses, four or five times daily, have been used with success. Marked cardiac weakness with disturbance of rhythm has been the indication for it; where there is any mural degeneration it is not sufficient.

Convallaria is a cardiac tonic of doubtful action; it sometimes succeeds where *digitalis* fails, but it evidently has a narrow sphere of usefulness. As it augments the contractile force of the heart, it aids diuresis. When mental and nervous symptoms are conspicuous, it may be indicated. In mitral stenosis, when digitalis is contraindicated, it may be serviceable in the tincture (m. 5-10) three to six times daily. *Convallamarin,* the glucoside, may be employed in half grain doses two or three times daily. *Adonis vernalis* has been a useful remedy in my hands. It properly belongs to the milder cases of failing compensation and it does not show any decided results until it has been given for some time, hence many have condemned its use. In conjunction or in alternation with *digitalis* or *strophanthus* it may be given in ten to fifteen drop doses of the tincture every three hours in grave cases. In cases convalescing from compensatory failure, it is decidedly valuable. In this respect it supplements or succeeds the stronger tonics. *Adonidin,* the active principle, may be given in 1/6 to 1/3 grain doses three or four times daily.

Agaricin is a remedy which has been strongly advocated by Goodno. He claims its greatest efficiency may be found in "cases of extreme dilatation of the right heart secondary to mitral disease or emphysema of the lungs." Two or three grain doses of the first decimal trituration, repeated every one to three hours, gives more than temporary relief. It is claimed by the same authority that it can not be excelled as a remedy in cardioplegia. Troublesome perspiration and neurasthenic excitement are suggestive symptoms. I have also found it very useful in the higher potencies in cardiac diseases in nervous children. *Cactus grandiflorus* is also of some value as a cardiac stimulant. Its action is best in small doses of the tincture and should be given in accordance with its indicating symptoms. *Crategus* has been advocated recently as a heart tonic. In the early stages of compensatory failure it augments the force of the heart's contractions decidedly; even in the later stages it may display its stimulating power. It has a powerful action upon the pneumogastric, correcting its inhibitory function when cardiac failure exists as a result of too much sympathetic stimulation.

Accessory heart tonics are useful agents in accordance with the demands of the symptoms and conditions. In this respect we may call into use many remedies and adjuvants which emergency and symptomatology may suggest. For instance, *morphin* may be regarded as a heart stimulant. The dyspnea or cardiac asthma which so often occurs at night, when the patient is in a state of nervous exhaustion, is greatly relieved by this remedy. Its hypodermatic use is best, 1/10 to 1/4 grain doses being employed. With *atropin* (gr. 1/150) added, the quality of the pulse will be improved and the respiration will be greatly relieved. The stimulating effect of the remedy given by mouth is not so pronounced. Under the same head we may place the use of hypnotics. Generally speaking, these agents should not be employed. There are times, however, when cardiac quietude is obtained only when we force the nervous system to rest. Though fearing a possible resulting depression from such measures, I am positive that I have by this means many times helped a patient out of a dangerous condition. *Chloralamid* (gr. 10-25) has been my favorite prescription for this purpose; it has not the depressing action of chloral hydrate and the stomach will tolerate it if it is given in cool water. *Trional* (gr. 15-30) in hot water, *sulphonal* (gr. 10-15) or *passiflora* (m. 30-60) may be preferred. In fact, there are many other milder measures which obtain hypnotic effect, and these should not be disregarded. Any safe remedy which insures nerve rest and does not depress the heart too much may be countenanced.

In addition to these suggestions for the heroic management of cardiac failure, we must remember that these and other remedies must be studied in accordance with their symptomatic action. Whether any one remedy in potency and truly indicated by its physiologic symptoms, will suffice in this contingency, must be answered by every physician in accordance with his experience and belief. I regard the stage of failing compensation as an emergency which demands prompt and vigorous treatment. Any other treatment will, to my mind, permit cardiac dilatation to reach a degree which can not be overcome. The study of the following remedies from the homeopathic standpoint should be of guiding influence in our practice. *Aconite,* though a dangerous cardiac depressant when used in the tincture, may, in the third decimal potency, arrest the cardiac arrhythmia which predisposes to failing compensation. As it lowers the arterial pressure and subdues nervous excitement it may be a powerful expedient in the early stages. We may compare this with *belladonna, cimicifuca, coffea* and *convallaria. Veratrum viride,* with its more decided action upon the cerebro-spinal system will give better control of arterial excitement, and may have a more protracted effect in cardiac weakness. It is in this connection that we must study the effects of *cactus grandiflorus* in its continued use. This, too, acts upon the circular fibres of the heart and arteries, the sensation of constriction being a guiding symptom. *Kalmia latifolia* refers more particularly to the heart palpitation with oppression in the breathing. It is indicated during the hypertrophic process following rheumatic fever, in which shooting and stabbing pains are quite constant. *Bryonia* and *spigelia* simulate this in many respects. *Lilium tigrinum* has more of a dull and pressive pain in the precordial region and it is related to other symptoms pertaining to the generative organs. *Collinsonia* is sometimes indicated when the cardiac excitement is induced by portal disturbances. *Colchicum* has many heart symptoms attending rheumatic and arthritic conditions; and *gelsemium,* like *rhus tox, baptisia* and similar remedies will often answer for the cardiac pains and perversions which follow acute infections.

In the *nervous influences* which create or complicate cardiac disturbances, we shall find many remedies to choose from. *Ignatia* in the intense nerve excitement of hysterical affections; *cannabis sativa* in excessive reflex irritations; *cannabis indica* in the peculiar mental perversions; *melilotus* in the congestive headaches from nervous causes are all a part of our armamentarium. When tachycardia is a feature, we shall invariably find help from such remedies as *lycopus, fucus, valerianate of ammonia* and *asafetida.* The degenerative conditions will call for remedies like *argentum nitricum, arsenicum iodid, iodonucleoid, aurum* or the *iodids* of *strontium* or *potassium.*

Special symptoms will demand treatment in accordance with a particular symptomatology or well known expedients. For instance, anemia will demand consideration in line with previously mentioned principles. Cough is so often a disturbing symptom that remedies like *phosphorus, ammonium mur., hepar sulphur* or *tartar emetic* must be used in accordance with their indications. Very frequently, however, this is due to hypostatic conditions depending upon cardiac inadequacy and as soon as the heart is relieved this trouble disappears. Hemoptysis, if not helped by cardiac stimulants, may demand such remedies as *secale, ipecac, nitric acid* or *suprarenal extracts.* Palpitation of the heart and persistent pain will often respond to the use of the icebag or remedies referring to this particular feature. The cause of this complication must be carefully looked for. These attacks may be anginal, and thus the treatment will be in accordance with directions under that subject. Often gastralgia is the irritating factor and other reflex causes may be regulated by symptomatic treatment.

The Treatment of Dropsy.—In valvular diseases to relieve the condition of dropsy will often tax the physician's ability to the utmost. It is an indication of cardiac inadequacy and the consequent obstruction to the capillary circulation. As the transuded serum further hinders the systemic circulation, the cardiac embarrassment may increase until it becomes an obligation to relieve both at the earliest possible moment. To this end we must push the heart stimulants to the point of physical endurance. Unless prompt relief is given to this condition, the heart will completely fail in its function and it is only a question of time when the renal excretion will be decreased to the danger limit. Palliation and trifling remedies will not suffice in this stage. The drugs mentioned as cardiac stimulants must be pushed to their physiologic action. In this respect *digitalis* and the compound tablet mentioned before is our best resource. The other heart stimulants may give additional help.

Purgative assistance is an essential requirement. If the salines and the increased use of distilled water do not establish thorough diuretic action, a more active cathartic will be required. *Cascara,* and its various compounds, or possibly the action of *calomel,* may be required. Elaterin (grs. 1/16-1/10), one pill three times daily, causes an efficient catharsis. *Diaphoretics* and the use of such drugs as *pilocarpin* are nowadays less employed on account of their serious reactions. The use of the trocar should be only a last resort, as it is apparent that the moment this procedure is started it must be a constant resort. *Multiple incisions* through the skin of the ankles will generally encourage the drainage of sufficient serum to give great relief to the dropsy and the renal insufficiency. I have seen some of the most intractable cases promptly relieved in this way. This method often gives more than temporary relief. Caution must always be observed lest the wounds become infected. I am happy to state that in my recent experiences I have abandoned this procedure entirely, as I have found it possible to accomplish the same results by a sufficient cardiac stimulation. I have found many times, when digitalis has failed me, that the best diuretic action was obtained from the internal use of *diuretin.* This has been given in as large doses as 40 to 60 grains in twenty-four hours. In my experience, however, best results have come from its use as an adjuvant to *digitalis.* In such a procedure I give the usual dosage of the latter remedy and I give, additionally, to adults 10 to 15 grains of *diuretin* every three or four hours, according to the conditions of the case. Thus far I have had no bad results from its use. The greatest objection is its taste, but this may be covered by adding a dram or two of essence of pepsin. The *acetate* of *potash* may also be of adjuvant service in this respect, but it does not yield the quick results which are obtained by diuretin. Two of the newer remedies have been of greatest service. These are *hydragogin* and *anasarcin*. The latter I have used but little, yet many of my associates speak of it with highest praise in cardiac dropsy. *Hydragogin* is, in my experience, one of the best remedies ever used for this purpose. It is given in 10 to 15 drop doses every three hours. In the last year I have obtained permanent relief of dropsy with this remedy in several cases. Fortunately, a majority of these cases are still under my observation, and I am happy to say that the chronic cardiac disease in each case is now being gradually helped by the indicated remedy after the *hydragogin* has done its heroic work. To me it is a great relief to find measures for this purpose outside of the unsatisfactory use of the trocar. *Hydragogin* is a combination of *digitalis* (1.5 parts), *strophanthus* (2.5 parts), *scillipicrin* and *scillitoxin,* which are the active principles of *scilla maritima,* and *oxysaponin,* which is obtained from *herniaria glabra.* The diuretic effect is particularly enhanced by the *oxysaponin.* This compound acts directly on the cardio-vascular system, kidneys and bowels. When *hydragogin* seems to be insufficient as a diuretic for very severe cases it will be best to supplement its action by the use of 1/60 grain of *strychnia* every three hours in alternation. The relation of cardiac dropsy to renal insufficiency naturally makes this phase of cardiac disease of vital importance in the consideration and treatment of kidney diseases. As we stimulate the heart's action we encourage a healthy renal circulation and this is the first essential in the treatment of those diseases. If the time would allow I might relate several severe cases of nephritis which in my care are progressing toward an eventual cure by the aid of this principle of cardiac stimulation. And the remedy which has been of greatest service in this respect is the *hydragogin* mentioned above. It is therefore my prediction that the time is coming when we shall master such apparently hopeless diseases as nephritis through a better appreciation and application of cardiac stimulation.

CARDIAC HYPERTROPHY.

By this term is meant an increase in the cardiac muscular substance. It is usually limited to the walls of one or more chambers, the ventricles being involved oftener than the auricles and the left more frequently than the right. The heart as a whole may be hypertrophied, the process of muscular increase extending by degrees from one chamber to another. Dilatation is frequently associated with this.

Etiology.—Hypertrophy of the muscular tissue of the heart occurs physiologically as the result of excessive functional activity. It is a normal process in a sense so long as the tissue is healthy enough to resist dilatation. When degeneration of muscular fibres exists, the physiological demands on the heart can not be maintained by this hypertrophy. In other respects it is a healthy compensating increase in the heart wall in order that it may endure excessive work. It is an idiopathic condition in certain occupations which demand excessive physical exertion. Extreme distention of blood vessels, as observed in beer drinkers, and the excessive use of alcohol, coffee, tea, etc., predispose to it. Functional nervous diseases incite the heart to overaction, while athletic excesses contribute to the same hypertrophic increase. Arteriosclerosis is a favorable pathologic state for its development following any excessive heart strain. A congenital hypertrophy is a possibility in rare cases. The left ventricle is enlarged from obstructive valvular lesions, insufficiencies, fibroid myocarditis and pericardial adhesions. Aneurysms, general arteriosclerosis and increased arterial pressure from contraction of the peripheral vessels accompanying nephritis, lead poisoning, gout, syphilis, etc., are common causes, since they induce increased cardiac activity. Hypertrophy of the right ventricle follows any obstruction to the pulmonary circulation or any perversion of the circulation through the right ventricle. Emphysema, bronchitis, lung collapse or consolidation will obstruct the natural pulmonary circulation and cause thickening of the walls of the right ventricle by its over activity. The right-sided valvular lesions will also lead to the same results. Hypertrophy of the left auricle will follow the auricular dilatation dependent upon mitral stenosis and, more rarely mitral insufficiency. Hypertrophy of the right auricle is a sequence of increased blood pressure in the pulmonary vessels and stenosis of the tricuspid.

Pathology.—Hypertrophy without dilatation is the simple variety in which the mural thickening is attended by no increase in the size of the cavity. Hypertrophy with dilatation is the form in which the increased size of the cavity, together with the enhanced thickness of the walls leads to the development of an unusually large heart. The muscular increase is due to the addition of new fibres and not to changes in the tissue structure. The connective-tissue increase, which has been described by some authors, is considered as a false hypertrophy.

The heart is increased in weight and size. The weight may rarely attain the extreme of thirty or forty pounds and the contour will vary as the hypertrophy affects different chambers of the heart. When the left heart is invaded, the organ will be elongated, conical and extended to the left. When the right heart is the seat of hypertrophy, the enlargement is to the right, the heart assuming a spherical shape. When both ventricles are enlarged, the apex becomes broader and in pronounced enlargement of the right ventricle, the left ventricle is drawn away from the chest wall and the right ventricle will virtually define the apex. The papillary muscles and columnae carnae are greatly thickened, particularly in the form attended by dilatation. The muscular structure has a deep red color and is very firm. As the muscular fibres increase, the heart sinks somewhat lower in the chest cavity. Other pathologic changes will depend upon the complicating valvular lesion which predisposes.

Symptoms.—The symptoms of cardiac hypertrophy are rarely suggestive unless there is a certain amount of failing compensation, when they may be general or local. The general symptoms are observed as the result of excited cerebral circulation incident to the perverted cardiac function. Among these we shall notice headache, tinnitus aurium, carotid pulsations, hot flushes and vertigo. Exophthalmos and epistaxis are also common symptoms following the development of the hypertrophy. The local symptoms will be observed first as a sense of precordial discomfort when the patient lies upon the back or left side. Pain and palpitation are observed only in proportion to the attending nervous symptoms. Active exercise or violent emotion will increase the characteristic symptoms. Arteriosclerosis and endarteritis are secondary conditions which commonly follow the high arterial tension incident to the hypertrophy. Apoplexy in the brain or lungs, as well as hemoptysis may be attendant features.

Physical Signs in Left-Sided Hypertrophy. —*Inspection* shows, particularly in children, a precordial prominence and an extensive visible impulse. The cardiac action is occasionally so exaggerated that the impulse is transmitted over the whole body, even causing the bed upon which the patient lies to shake. *Palpation* discerns the slow, forcible and heaving character of the impulse. This will be more abrupt when there is dilatation. The apex beat is displaced downward as far as the sixth intercostal space; sometimes it reaches the seventh or perhaps the eighth interspace and may extend to the left of the mammillary line from one to three inches. A diastolic impulse is occasionally felt in the aortic interspace. The pulse is strong and regular, except in association with dilatation, when it is more abrupt, soft and somewhat accelerated. *Percussion* discloses a dulness extending downward and to the left. *Auscultation* detects the loud, prolonged and booming character of the first sound which will be shorter and sharper when dilatation complicates. Aortic accentuation and second sound reduplication are infrequently associated; the second sound will, however, be less intense in cardiac dilatation or weakness. Valvular defects when they coexist will change these murmurs.

Physical Signs in Right-Sided Hypertrophy.—*Inspection* shows a bulging of the lower sternum and the sixth and seventh left costal cartilages; there will also be a visible and diffuse impulse over the same area which may extend to the epigastrium. In case of much dilatation when the chest wall is thin, a similar pulsation may be seen in the third and fourth interspaces to the right of the sternum. *Palpation* finds the apex beat carried to the left, diffuse and but slightly displaced downward. *Percussion* defines an increase in cardiac dulness an inch or more to the right of the sternum. *Auscultation* presents but little of value in the simple hypertrophy. The first sound over the tricuspid area is somewhat louder than in the normal heart and the pulmonary second sound is accentuated on account of the increased tension in the pulmonary artery. A reduplication of the second sound may be heard. Associate dilatation will magnify these features. Emphysema, however, may occlude or pervert these sounds.

The *symptoms* following this hypertrophy of the right heart should be taken into consideration. On account of the augmentation of the tension in the pulmonary vessels, *dyspnea* will be in evidence as a result of undue exertion or excitement. When the hypertrophy follows emphysema or interstitial pulmonary obstruction, the symptoms of precordial distress, cough and dyspnea may mask other symptoms. When dilatation and relative tricuspid insufficiency ensues, dyspnea, bronchitis, pulmonary edema, cyanosis, hemoptysis and systemic venous stasis will remain as permanent symptoms, unless some relief is instituted.

Physical Signs of Auricular Hypertrophy.—Accurate physical signs for these pathologic conditions are not so well defined or easy for discernment as in ventricular enlargement. The left auricular hypertrophy may disclose some dulness in the left second or third interspaces, and a presystolic wave may be noticed at the second interspace of the same side. When the signs of mitral stenosis or mitral insufficiency are found, we may be more positive that hypertrophy is present here. Hypertrophy of the right auricle is invariably attended by dilatation of the same. It is a condition secondary to stenosis or insufficiency of the tricuspid valve. Dulness will be found in the third and fourth interspaces to the right of the sternum. A presystolic pulsation may be discovered in the same area. Systolic jugular throbbing and signs of general venous engorgement are confirming symptoms.

Diagnosis.—The physical findings must be supported by the predisposing causes to arrive at a satisfactory diagnosis. The following differentiations must be made: *Fibroid phthisis* with retraction, *interstitial pneumonia* and *chronic dry pleurisy* affecting the left lung, may expose so much of the right side of the heart that they may simulate hypertrophy of the heart. The evidence of pulmonary disease, the limited lung expansion, the displacement of the heart upward and to the left, together with absence of the heaving impact, should determine the difference. *Aneurysm, pericardial effusion* and *mediastinal growths* simulate hypertrophy by the increased area of dulness, but the lack of a forcible and heaving impulse, together with other positive signs of hypertrophy, should settle the doubt. *Cardiac hypertrophy* arising from the tachycardia of neurasthenia or exophthalmic goitre, will be easily differentiated when the typical and confirming symptoms of such diseases are taken into careful consideration. *Displacement of the heart* ought to be recognized by the chest malformations and the absence of the positive physical signs which characterize true hypertrophy.

Prognosis.—Physiologic hypertrophy which represents normal compensation is not to be regarded in an unfavorable light. The functional enlargement which attends neurotic conditions is also amenable to the treatment which relieves the cause. When it exists as a sequence of organic cardiac lesions or diseases of the blood vessels, the outcome is less favorable. There is decidedly more danger in hypertrophy of the right heart as this leads to obstruction in the pulmonary circulation, which is always serious. When dilatation succeeds hypertrophy we shall also expect unfavorable results. Degeneration of the cardiac muscle which reduces nutrition indicates a very grave prognosis. In every case much depends upon the contributing cause, the age and the natural physical resistance of the patient.

Treatment.—In the treatment we must consider the habits of the patient and the general nutritive condition. In every case the aim must be to maintain a proper compensation of the cardiac muscle. No unnecessary nervous excitement or muscular strain must be tolerated. A *quiet life* is an absolute necessity. If the occupation favors cardiac strain, a different vocation must be adopted. *Stimulants* and *overeating* must never be indulged in. Nutrition must be maintained by every means which favors that end. *Outdoor life* and a proper amount of rest will be favorable contributing factors. The *diet* should be nitrogenous as much as possible, unless a kidney complication forbids. Liquids should constitute only a small part of the food principle and digestive disturbances should receive prompt remedial and dietary relief. If *constipation exists,* it must be corrected by a regulated diet, the natural indicated remedy or, if necessary, by a mild saline cathartic. Cathartics are needed only in extreme cases. *Exercise* should be instituted in such a manner as to encourage a natural physiologic activity of the cardiac muscle. Violent athletics will not do this, but a systematic and careful use of some of the various exercisers advertised or light dumb-bell practice will help the ordinary case. *Warm baths* for their mild sedative effect and the morning cold sponge will give tone to the heart muscle as they add strength to the general system.

The *organic heart lesions* which lead to essential hypertrophy should be treated in line with the recommendations given in the chapter on valvular diseases. Any of the remedies mentioned there should be employed as indicated. Cardiac tonics are to be used only when they are necessary to regulate the abnormal heart action; their overuse will cause an equally dangerous reaction. The systemic venous stasis, which exists when the right heart is involved, will naturally be modified by measures employed for the relief of portal congestion. It is not always necessary in these cases to resort to a hydragogue. Such remedies as *iris versicolor, chelidonium, chionanthus* and *podophyllum* may answer for this purpose as their gradual use will restore a normal hepatic and intestinal action. *Chinoidin* in two grain doses will often regulate the hepatic congestion and it need not be given very frequently. An occasional teaspoonful of granular sodium phosphate will in many cases be sufficient. In more obstinate conditions it may be wise to administer quarter or half grain doses of *mercurius dulcis,* but this should be avoided if possible.

It must be remembered at all times that no remedy or treatment may be employed which in any sense interferes with the progress of compensating hypertrophy. This is a natural process which serves as a cardiac protection. For this reason internal remedies must be employed with care. The factors which lead to this condition should receive most attention. Diseases of the lungs which may precede or attend must be managed in accordance with suggestions under that topic. Such remedies as *phosphorus, bryonia, hepar sulphur* and *tartar emetic* will be of unusual value when the pulmonary symptoms call for them. Remedies which act as vaso-dilators may be of service when the cause can be traced to arteriosclerosis. *Glonoin* and *veratrum viride* will be thought of in this respect. Their continued use, however, should be watched with care. Symptoms pertaining to the nervous system demand remedies which have been found useful in that sphere.

Medicines employed for the *relief of symptoms* will tend to control the progress of the disease, and this is of paramount importance. The homeopathic principle applies here with telling effect. *Aconite* is first thought of, it having the symptoms of nervous palpitation, dry skin, extreme restlessness, etc., which are so frequently present. When it is given for these cardinal expressions it sustains the cardiac rhythm. If it is employed too long and in low potencies, its cumulative action will thwart the object of its use. *Coffea, caffein, tabacum* and *hyoscyamus* may be compared. *Pulsatilla, ignatia* and *cimicifuga* may, in certain cases, have similar indications. *Belladonna* and *stramonium* will control many of the distressing nervous and cerebral symptoms. *Calcarea, silicea, sulphur* and the *ferrums* will be useful in nutritional deficiency, particularly in young people. *Rhus tox, bryonia* and *spigelia* can be depended upon for the symptoms enumerated before. *Arsenicum* as a standard cardiac tonic should never be overlooked, and *lachesis* has a similar usefulness in later life. Among the common homeopathic remedies *cactus grandiflorus* covers many of the symptoms of abnormal cardiac activity. A feeling as if the heart was strongly grasped by a heavy hand, attended by palpitation and the exhausted state, will call for it. *Lilium tigrinum* has many similar symptoms, but a dull pressive pain in the precordia and the constant dull frontal headache are most characteristic. *Kalmia* corresponds to the rapid heart action with a tendency to weakness and dyspnea. *Digitalis* in the third decimal potency covers a majority of these symptoms and may be used in this strength for a long time with results which are not always obtained by the larger doses. For the symptoms of heart hurry we may safely rely upon such standard remedies as *lycopus, fucus, valerian, asafetida, moschus* and *pulsatilla*. *Naja, crotalus* and *apis* will demand due symptomatological consideration.

CARDIAC DILATATION.

Dilatation of the heart refers to an enlargement of one or more of its chambers. This may present three pathological conditions: (1) Simple dilatation in which the walls are of normal thickness. (2) Dilatation with hypertrophy. (3) Dilatation with thinning of the wall. These three forms may be variously combined.

Etiology and Pathology.—Increased pressure within the cavities and diminished resistance of the walls from muscular weakness or disease, acting singly or together, create dilatation. The increased pressure depends on augmented systemic or pulmonary blood-pressure or on some obstruction which perverts the natural rhythmic flow through the heart chambers. These causes and their pathology have been mentioned under the subject of hypertrophy. So long as the hypertrophy properly compensates, dilatation is of little consequence, but when this fails, the enlargement of one or more chambers is proportionate to the pressure, the obstruction and the weakness of the walls. An abrupt and marked increase of the pressure, which follows violent muscular effort, is a frequent cause. Diminished resistance comes from myocardial weakness. This is a natural sequence of endocarditis or pericarditis following infectious diseases. Fatty and fibroid degeneration of the muscle favor it because of impaired contractile power; in the same way nutritional diseases predispose by the resulting enfeeblement of the cardiac muscle. Diseases of the blood may be considered under this head. A gradual dilatation will sometimes ensue as the result of persistent irregularities in diet, and the use of alcoholic stimulants and tobacco. Neurotic causes furnish a few cases. Idiopathic dilatation is rarely observed. The athlete and the bicyclist are often afflicted with cardiac hypertrophy and subsequent dilatation.

The walls, which are subnormal in tone, will yield most easily to the increased tension. This depends very much upon the valvular lesion which causes the irregular flow of blood through the ventricles. A mitral stenosis or insufficiency leads to left auricular and right heart dilatation, while aortic insufficiency or stenosis may, with the addition of a mitral insuffiency, permit a left ventricular dilatation. Increased pulmonary tension naturally favors dilatation of the right ventricle, and the auricles yield easily on account of their thin walls.

Symptoms.—The symptoms depend upon the degree of pressure, the condition of the cardiac wall and the part of the heart involved. When dilatation takes place slowly, the signs of gradual heart failure will be recognized. Everything depends upon the compensating action. Acute dilatation will give signs of sudden enfeeblement. *Palpitation* is an early symptom, as it is expressive of the compensatory failure and the beginning of arrhythmia. *Dyspnea* appears with the slightest exertion as the pulmonary stasis increases. On this account the patient often finds it impossible to lie down. *Precordial pain,* a rapid, weak and intermitting pulse, are sometimes disturbing symptoms. *Cyanosis,* disturbances of the nervous system, coldness of the extremities, albuminous and bloody urine, and pulmonary involvement follow in natural sequence.

Dropsy is a pathognomonic sign of cardiac failure. It begins in the extremities and extends upward invading the genitals. The face also becomes edematous and general anasarca may result as compensatory protection becomes weaker. Hypostatic congestion affects the liver and other organs, and the brain may in the later stages suffer severely from this cause.

Physical Signs.—*Inspection* shows a widely diffused, wavy and undulating impulse. The apex beat is displaced downward and outward and is not easily located. *Palpation* confirms the feeble and diffuse character of the impulse, though there are occasions in which palpation gives negative results. This has been regarded as a clinical sign worthy of note. The pulse is small, short, rapid and irregular. *Percussion* reveals dulness extending to the left and downward, varying with the degree of dilatation. When the right ventricle is greatly affected, the transverse dulness is increased; vertical dulness is more pronounced in case of auricular enlargement. *Auscultation* reveals the marked feebleness of the sounds. The first sound is short and flapping, while the second sound may be indistinct. Both sounds may be reduplicated. A galloping rhythm is distinctive. The murmurs of valvular lesions may interfere, but these are inclined to vanish suddenly or be confused with other sounds, which is indicative of the cardiac weakness.

Diagnosis.—The diagnosis is made upon the following clearly defined symptoms and physical findings: A weak and irregular heart action, with a diffuse and undulating impulse; a small, short, irregular and rapid pulse; an indistinct apex beat; the outward and downward, transverse or vertical increase in percussion dulness; a short and flapping first sound, with a weakened second sound and the galloping rhythm. Differentiation must be made in the case of *hypertrophy*. This, however, has a slow, strong and heaving impulse, a distinct and rounded apex beat to the left and downward, with a loud and prolonged first sound, followed by an accentuated second sound.

Prognosis.—In general the result is unfavorable, but it depends upon the causative conditions and the degree of compensatory hypertrophy. *Myocardial degeneration* favors a fatal termination and a recurrence should be regarded with gravest fears. *Pulmonary edema* and persistent general dropsy are always danger signals. If, however, prompt and careful treatment is applied, many cases may be safeguarded against further dilatation. Interstitial nephritis makes the occurrence of cardiac dilatation a serious matter; the resistance which has existed in the renal circulation for any length of time, as occurs in such diseases, favors a gradual yielding of the cardiac walls which can not well be compensated.

Treatment.—The first principle of treatment must consider *absolute rest* and freedom from exertion and excitement. The patient must assume at once a recumbent or semi-recumbent position. To obtain this a physician must often thwart the efforts of friends who seem to think it necessary to keep the sufferer bolt upright and excited as much as possible. The recumbent posture in the beginning will often do more for compensation than remedies. The heart muscle at first will tend to contract if all other resistance is taken away, and if respiration is easy the venous blood is brought to the right heart less rapidly. The next early necessity is to *relieve the portal congestion* by mild and yet thorough catharsis. Salines may be sufficient in some cases. I believe in the average case of dilatation that the administration of *digitalis* is indicated. It certainly regulates the heart's action promptly, as no other remedy will do. Whether it should be continued is a matter of individual judgment. Ten to fifteen drops of the tincture should be used for adults, at intervals of four to six hours. It may be administered oftener in emergency. Younger people may not tolerate such heroic doses. The infusion may be used in two dram doses if it is properly made from the green leaves. Digitalin (gr. 1/100) often acts better hypodermatically. The other standard heart stimulants, *adonis, convallaria, apocynum, strophanthus, strychnia, morphin,* etc., may be employed as seems best when digitalis acts unfavorably or disturbs the stomach.

It is often a mistake to continue cardiac stimulants of this kind too long and, after the heart has been quieted it will often be better to use a well indicated remedy and insist upon absolute quietude, thus enabling the heart to assume a more natural compensation. *Diet,* which must at first be limited, should later be more nourishing in character in order to obtain a nutrition which will properly sustain the failing muscular power. This is too often overlooked in the chronic stage. Excitement and exertion must be watched with greatest concern. As the *chronic state ensues,* massage, faradism, baths and appropriate muscular exercise will be useful but should be used with greatest caution. If the patient is in the least irritated by the measures, they should be discontinued. So few physicians appreciate this that emphasis on this point must be made. The *Nauheim baths,* which have lately become a fad, are efficient in some cases, but should not be resorted to indiscriminately. In acute dilatation they are not always safe. In the chronic period they may be efficacious. ***Resistance exercises*** should be considered in this same category. There is no doubt that in the chronic state this method will enhance the contractility of the heart muscle; still the danger of over-exercise must always be guarded against. ***Blood-letting*** still has its advocates when the right heart is surcharged. The *icebag* may be employed successfully in many cases. We must keep in mind that it is of paramount importance to watch the minutest detail of the patient's daily life to minimize the strain and excitement which are bound to attend. For this purpose we must delegate our control only to the assistance of a competent nurse.

The internal remedies which are naturally indicated and much of the general treatment have already been outlined under the head of valvular cardiac diseases.

MYOCARDITIS.

We speak of myocarditis as an inflammatory affection of the cardiac muscle. It occurs in the acute and chronic form.

ACUTE MYOCARDITIS.

Etiology.—Pericarditis and endocarditis following rheumatic fever are the most frequent causes. It is believed also that myocarditis may be a primary disease with the rheumatic specific as the direct cause. Infectious fevers and infectious emboli invading the coronary arteries are prominent causes. Ulcerative endocarditis, therefore, is naturally a contributing factor. The male is said to suffer from this disease more frequently than the female.

Pathology.—This relates to three distinct varieties. *Acute parenchymatous myocarditis* is a granular degeneration affecting the muscular fibres of the parenchyma. In this we find a soft and very pale looking muscle structure with marked increase of nuclei. *Acute diffuse interstitial myocarditis* is characterized by a round-cell infiltration. *Acute circumscribed myocarditis* is the most common variety recognized. In this the degenerative process is characterized by an abscess formation which generally covers a limited area. While there is a tendency to circumscribed inflammation, the abscesses are yet liable to occur in the multiple form with rupture into the pericardium or into the cardiac chambers. Thus pus may enter the circulation through this fistulous opening into the heart, which accounts for remote embolic effects. Rupture into the pericardium or aneurysmal dilatation of the heart may ensue as the blood pressure distends the abscess cavity. The gradual yielding of the muscle attends the regular ventricular diastole. The mitral or tricuspid valves may be the seat of this process and eventually lead to insufficiencies which are very dangerous. The septum in rare cases has been perforated, allowing a direct communication between the right and left heart. Caseous and calcareous degenerations are ultimate possibilities. The left ventricle is affected most frequently.

Symptoms.—The predisposing disease is often apparent, and the symptoms which go with it may be reflected in the acute myocarditis. The first signs are usually suggestive of cardiac enfeeblement. *Shortness of breath,* palpitation, vertigo and sudden syncope may unexpectedly appear in the course of the infectious disease which is so frequently the cause. The *pulse* is irregular, rapid, small and soft. *Cyanosis* and pulmonary congestion will appear in the later stages. *Cold sweats,* diminished urine and delirium are symptoms of the later periods of the septic infection. The *nervous symptoms* are often similar to those of meningitis.

Physical Signs.—The cardiac activity indicates enfeeblement; it is tumultuous and irritable and the sounds are sharp and short. The murmurs are dependent upon the valve involved and the extent of its lesion, as well as the amount of cardiac dilatation following the degeneration. The conjoint signs of embolic infection depend upon the viscera involved, but the septic type of fever is invariably present. The *diag-*

nosis requires a perfect understanding of the pathology and the symptoms enumerated above. The *prognosis* is fatal in diffuse forms, but the circumscribed forms may yield to treatment. Sudden death is the natural termination.

Treatment.—*Rest* is a necessity to sustain the heart against any rupture of the cardiac wall. *Stimulating remedies* should be used only as a last resort. Whiskey in moderate doses may be tolerated. *Strychnia* may be demanded in some cases. *Arsenicum iodid, nux vomica* or *ignatia* in the second decimal potency may furnish sufficient stimulation. The following remedies may be employed in accordance with their indications: *Aconite, arsenicum, baptisia, echinacea, muriatic acid, iodin, iodoform, lachesis, naja* and *phosphorus.*

CHRONIC MYOCARDITIS.

Etiology.—Chronic myocarditis may be due to extension of the inflammatory process from endocarditis or pericarditis or, in rare instances, it may exist as a sequela from the acute form. Alcoholism, gout, rheumatism, malaria, diabetes, syphilis and chronic nephritis are regarded as predisposing causes. Typhoid, scarlet fever, diphtheria and influenza are also looked upon as possible agencies. Occupations which induce extreme cardiac strain, particularly when coronary sclerosis exists, perverted nutrition from a defective blood supply, or even the sedentary habits among those who indulge in high living, are to be reckoned as causes. Males are more naturally affected than females, and those past middle life furnish the greater number of cases.

Pathology.—Fibroid degeneration of the interstitial cardiac tissue is the pathologic change which takes place in this disease. This results in a diffuse or, more frequently, a localized area of induration. The left ventricular wall, the septum and the papillary muscles are the usual locations of these changes. The size of the lesion depends much upon the predisposing cause and the condition of the cardiac structure. There may be small indurated spots or the sclerotic process may cover a considerable portion of the ventricular wall. The color varies from a grayish-white to a grayish-yellow appearance. A simple hyperplasia of the connective tissue may be the only degenerative sign, or, in other cases, there may be a marked increase of new fibrous tissue. Fatty degeneration may invade so much of the parenchyma that a progressive atheroma eventually affects the heart as a whole. Fat globules are liable then to replace the normal structure, until grosser lesions follow. In the senile heart the failing nutrition is liable to induce a combination of fibrous and fatty degeneration. Fatty overgrowth in obese patients may lead to such an increase of myocardial fat that the fibres are transformed to the degree that atrophy ensues. Thrombosis or embolism of the coronaries creates a localized anemia of the cardiac muscular tissue, and this part subsequently undergoes a sclerotic change. Such a condition may follow a general sclerosis of the arteries, as well as the more frequent obliterating endarteritis. Cardiac hypertrophy is a common sequence of the more extensive arterial hardening, though a compensatory hypertrophy of the uninvolved part of the cardiac muscle may develop at any time. Dilatation and valvular incompetency are more serious results which often appear as the sclerosis weakens the heart wall or destroys the function of the papillary muscles.

Symptoms.—The mild cases, and in many instances the severer types, often show no distinctive symptoms. This may be due in part to a compensatory hypertrophy which masks the pathologic signs by its protection of the heart function, or it may be explained by the fact that the patient is in a good state of physical preservation. Evidence of fatigue from slight exertion, or other indications of cardiac failure, may be regarded as the earliest suggestions of the disease. We observe it most frequently in men beyond middle life who have lived a sedentary home life, but an active business life. With such subjects there is an alarming tendency to overeating and drinking. The abnormal blood-pressure in such patients is overlooked and the troublesome shortness of breath is charged to the increase of fat which comes from such habits. The fatty increase in the heart muscle is not thought of except for the aortic second sound accentuation, which is due to high arterial tension and developing cardiac weakness. Another class of symptoms occurs in association with anginal attacks in which dyspnea, palpitation and a sense of precordial constriction give rise to much paroxysmal suffering. *General venous stasis,* which affects the liver, kidneys and intestinal tract and leads to marked edema, is a subsequent development. These features may be so pronounced that decided mental disturbances follow. *Pseudo-apoplectic seizures,* convulsive twitchings and often cerebral hemorrhage may be incidental to extreme physical exertion or incautious eating or stimulation.

Cardiac arrhythmia is characteristic of one class of cases. In these the pulse will be decidedly irregular and intermittent; for a time it is slow and strong and then again it becomes rapid and feeble; in certain instances its irregularity and character are so atypical that a safe opinion as to the condition can not be formed. Bradycardia generally predominates and the pulse-rate is reduced to fifty or even forty beats per minute. If cardiac atrophy exists the pulse is usually slow, irregular and of low tension. When fatty degeneration is present it is liable to be more rapid, though irregular. Tachycardia of a paroxysmal character is sometimes a distinctive symptom, and here the pulse rate may run above one hundred. An asthmatic form has been defined which is recognized by the semblance of asthma and the attendant pulmonary edema. The distressing dyspnea of this class of cases will be accompanied by an irritable heart action and a cough which simulates bronchial asthma. In senile cases and possibly in many others, stiff and tortuous arteries may be found and signs of interstitial nephritis will often be discovered. The Cheyne-Stokes respiration is sometimes a serious form of the dyspnea. In all cases, however, cardiac incompetence, the feeble or arrhythmic pulse and the signs of a developing systolic murmur must be looked for. The disease pursues a slow course, unless exciting causes endanger the heart's strength. The gouty diathesis may show itself in some patients and may often mask the true condition. Hypertrophy of the heart is also an incident in myocarditis when chronic nephritis leads to a circulatory disturbance which demands excessive cardiac activity. In this we shall observe other clinical manifestations which point to renal disease.

Physical Signs.—*Inspection* is negative in myocarditis. *Cyanosis* is observed in some advanced cases, and if the left ventricle is hypertrophied, the apex beat is displaced. Otherwise we shall notice the evidence of senile degeneration. *Palpation* locates the displaced apex beat when present and defines the character of the impulse, which is often not detected by the eye on account of its feebleness. By this means we also ascertain the condition of the arterial coats. Palpation of the liver is necessary to learn of the hepatic engorgement. *Percussion* is useful to outline the area of dulness, which varies in right- and left-heart hypertrophy. *Auscultation* shows a gradual change from the clear and strong heart sounds to those which are weak and muffled. If hypertrophy is pronounced, the first sound is prolonged, while the aortic second sound is accentuated, together with some intensification of the pulmonic second sound. The galloping rhythm will follow as dilatation develops.

Diagnosis.—The disease is characterized by the physical signs, plus the evidence of enfeebled cardiac action. It is necessary to exclude valvular lesions in making a correct diagnosis. Fatty overgrowth will be understood by its distinctive symptoms and history.

Prognosis.—The outlook in this disease may be considered as generally unfavorable. The predisposing causes and the associate conditions have much to do with the outcome. Arteriosclerosis, interstitial nephritis and diabetes are serious complications. The duration of the disease depends much upon the age, the general health of the patient and the past habits. A strenuous occupation can not be followed with safety in any stage of the disease. Occlusion of an arterial vessel may follow the sclerotic development. Sudden death is a possible termination, though a gradual extension of the cardiac enfeeblement is the more probable result.

Treatment.—The treatment does not offer much encouragement. It must be applied from the symptomatic standpoint. Generally speaking, we must observe the principles advised under the management of valvular lesions. *Rest* is a factor of greatest importance, and this must be absolute during the serious stages. *No excitement* of occupation or surrounding may be tolerated. This does not mean that a patient must be confined to the bed, as outdoor life is a necessity for the general health. *A mild climate* and the minimum of business cares are essentials of first importance. *Hygiene* should be maintained at all times and the diet must be limited to the most easily digested foods. *Alcoholic stimulants* can not be tolerated with safety, though, in small doses, they may be used as medicinal agents in elderly patients who have been accustomed to their use. Much depends upon the condition of the cardiac walls. If compensation is holding its own, there is a prospect of maintaining a safe chronic state. Such patients may indulge in a light form of exercise sufficient to prevent the over-production of fat. *Digestion* must be watched so that a proper assimilation is always maintained. The quality and quantity of food must not overtax the heart, the blood vessels or the kidneys. A mixed diet is always best; a vegetarian diet may induce an unhealthy obesity, while an extreme nitrogenous diet will always tend to produce high arterial tension. Gouty conditions will demand an abundance of pure water and other eliminative agencies. The Turkish bath is probably too severe in its reaction, and on the average we may depend more upon the morning cool sponge. Heart tonics are not usually indicated in this stage of the disease. The Nauheim system of baths is worthy of consideration in some cases.

When *compensation begins to fail,* we shall be obliged to resort to measures to preserve the contractile power of the heart. Here we may need such remedies as *ignatia, ammonium carb., hyoscyamus* or *cactus grand.* Possibly a more powerful stimulant will be called for, such as *digitalis, strophanthus* or *strychnia.* Digestion must now be watched more carefully than heretofore and if the portal system is suffering from stasis, it is well to administer a cathartic. Ordinarily, however, the indicated remedy and such agents as *nux vomica, iris, chelidonium* and *euonymin* in low potencies are sufficient for this purpose. *Nitroglycerin* (gr. 1/100) has been lauded for the reduction of blood pressure at this period, but its use must be undertaken with constant vigilance. *Erythrol tetranitrate* (grs. 1/2) once or twice daily, is also being successfully used as a vaso-dilator. *Potassium iodid,* the *chlorid of gold* and the *iodids of arsenic* and *strontium* are valuable agents for the same purpose. Food should be simple in character when compensation is weak, making it necessary to eliminate the heavy meats and limit the amount of water during the meals. Gentle exercise is a valuable adjuvant, but it must be considerately applied. Some have suggested cautious mountain climbing, but this is dangerous, in that it is likely to be overdone. Systematic exercise, obtained by walking, will give a healthy circulation to the heart muscle, and this is by all odds the safest expedient. The resistance exercises are also of service. No exercise should be indulged in to the point of fatigue. Tobacco and other excesses must be interdicted.

When *cardiac incompetence is extreme* we shall need a more vigorous application of the heart tonics mentioned before.

Digitalis (m. 10-15) of the fat-free tincture, should be given three or four times daily until the heart gives evidence of being supported. *Strophanthus,* tincture (m. 5-10), or *spartein sulphate* (gr. 1/10), or *strychnia* (gr. 1/60-1/40) may act better for this purpose in certain cases. The Nauheim baths have their greatest efficiency in this stage and the patient must observe enforced rest until there are signs of improvement in the cardiac tone, when limited exercise may be undertaken, but always with care lest there be a return to extreme incompetency. During this period a liquid diet is best. Another matter of importance is careful nursing. This should be entrusted to an experienced trained nurse rather than to the unsatisfactory nursing of excitable and exciting friends. Much harm is done by interested relatives at such a time as this. When arteriosclerosis exists we must be very cautious in the use of heart stimulants, particularly with strychnia. For the anginal attacks we shall get better results from the hypodermatic use of *morphin* (gr. 1/8-1/4). In severe cases it is best always to administer our heart tonics hypodermatically, though whiskey and aromatic spirits of ammonia may be used by mouth. When cardiac asthma intervenes, the hypodermatic use of *morphin* (gr. 1/8), combined with *atropine* (gr. 1/150), will give almost immediate relief. The dropsy and other symptoms following dilatation should be treated as outlined under that subject.

When such causes as syphilis, rheumatism and malaria predispose, we must go back to the remedies indicated in those diseases. *Mercury* and *potassium iodid* are still our best remedies in these cases. *Merc. corr.,* or *biniodid* are probably the best forms of the former and are to be used in the second decimal potency. *Cypridol* is a new preparation which is successfully used hypodermatically and internally. The *iodid of potash* gives the best results in the saturated solution. The rheumatic history will demand a careful study for remedies along that line, and malaria may call for the use of *quinin, china, eupatorium, natrum mur.* and other indicated remedies. Anemia is often a factor in this disease, and, therefore, *arsenic* is a standard remedy as a direct cardiac tonic. It often gives best results when combined with *china, strychnia* or *iron. Phos. acid, argentum metallicum, lachesis, naja* and *phosphorus* must be considered for the degenerative process in accordance with their clinical symptoms.

THE FATTY HEART.

Under this head we are to study two forms of fatty increase which, affecting the heart, change its function and size. (1) Fatty overgrowth or infiltration. (2) Fatty degeneration in which the heart muscle is to a great extent transformed into a fatty structure.

Fatty overgrowth or *infiltration* occurs in connection with general obesity. High living, overindulgence in fatty, saccharine and starchy foods, and excessive use of alcoholic beverages favor it. Sometimes it is an idiosyncrasy or inheritance. The fat is deposited beneath the pericardium, often forming a fatty envelope; infiltration between the muscular fibers is not uncommon and may extend even to the endocardium. This exerts a pressure which favors a more extensive metamorphosis.

Fatty Degeneration occurs secondary to the fatty accumulation, and also in connection with cardiac hypertrophy. It follows in the course of primary and secondary anemias, but it is most commonly found in connection with cachectic states attending such diseases as carcinoma and phthisis. Severe acute infections, as well as arsenic and phosphorus poisoning are occasional causes. Disease of the coronary arteries is a frequent factor in its production. Age and sex must also be considered, for we discover the disease usually after the fortieth year and more often in men than in women. Every influence which predisposes to defective nutrition of the muscle cells favors its development. The pathologic change is either localized or general, the most frequent seat being the left ventricle. At first yellow spots or stripes appear and these are soon transformed into a yellowish-brown color on account of an associate brown atrophy. The tissues become soft and friable and later oil drops and granules develop in the fibers, until finally the muscular striae and nuclei are transformed. The heart generally attains a considerable size. Besides the brown atrophy mentioned above, we may find other degenerative tendencies which partake of the calcareous, amyloid or hyaline type.

Symptoms.—The symptoms of fatty infiltration are not always uniform or suggestive of the real condition. *Dyspnea* and *precordial distress* are liable to develop with the slightest exertion. The *pulse* is feeble and rapid, but regular; vertigo, syncope, palpitation and cyanosis may appear. A passive bronchitis may be recognized and asthmatic attacks may attend this. The physical signs are not satisfactory inasmuch as the thickened chest wall makes auscultation unsatisfactory. The heart-beat is weak and its sounds are indistinct; the area of percussion dulness is increased.

The symptoms of fatty degeneration are also confusing. *Cardiac feebleness* with arrhythmia is a characteristic feature. Often the degeneration permits dilatation and then such symptoms as palpitation, dyspnea, a small irregular pulse and cold extremities may be observed. *Breathlessness,* an occasional retarded pulse, cardiac asthma and a semblance of angina pectoris following undue exertion, are later symptoms. *Intellectual disturbances,* which sometimes assume the form of maniacal delusions, pseudo-apoplectic attacks, Cheyne-Stokes respiration and epileptiform seizures are the more serious symptoms of fatty degeneration.

Diagnosis.—It is not always an easy matter to diagnose the presence of, or differentiate between, fatty infiltration and fatty degeneration. After excluding valvular diseases and dilatation, we may be reasonably sure of this disease when the symptoms of cardiac weakness described above are presented. It will be confirmatory evidence when the patient is past forty and carries an undue amount of fat.

Prognosis.—Neither fatty infiltration nor fatty degeneration respond well to treatment. The former, to be sure, is less liable to cause sudden death, yet the probability of a cure is minimized by the fact that it takes place during the later period of life. Everything depends upon the etiologic factors, the degree of degeneration and the probability of dilatation. Notwithstanding this, if the patient lives a cautious life and undergoes systematic treatment, much may be done to prevent a fatty increase and unfavorable complications. Sudden exertion, overeating and the use of stimulants must be completely prohibited. Any acute infection will increase the danger.

Treatment.—We must take into consideration every detail of the patient's physical peculiarities in undertaking the treatment of this disease. Where the tendency is toward obesity, we must correct it, if possible. When any habit which predisposes to this unhealthy increase is found, we must dethrone it. Sudden changes of habit are not best, but we must cut off the stimulants as soon as possible. The dietary must be adopted to accord with the individuality of the patient. Carbohydrates should be discriminated against on general principles, but the amount of fat and albumin to be consumed is a question hard to decide. While the aim is to encourage the absorption of fat, it is not always an easy matter to know how to regulate this in each case. Some patients seem to digest fats naturally, while others store it up in the tissues. The former, therefore, may need it, while to the latter it is harmful. A certain amount of albumin will stimulate the absorption of fat. As the heart muscle performs constant work, it is necessary to supply it with tissue-forming food such as proteids. We have noticed some of the most unfortunate results following a sudden reduction of obesity. Starvation is a dangerous process in this and all other diseases of the kind. It is possible to safely reduce the obesity by a careful and gradual reduction in the fat-forming foods. To this end the physician must constantly watch the patient and when the strength and weight are being reduced with signs of weakness, it may be best for a time to admit the albumin and starches in larger proportions.

Exercise is a valuable aid in promoting the oxidation of adipose tissue and it also institutes a healthy circulation through the heart muscle. This, too, must be undertaken with forethought. As overexercise creates fatigue, in some cases, particularly in the aged, massage may be best employed. *Catharsis,* obtained by the daily use of such waters as Vichy or Kissingen, may be resorted to with success, but even this method has been carried to such extremes that unfavorable depletion has resulted. In general we may say that a simple mixed diet will obtain a more natural assimilation.

As a proper support to these measures for the normal reduction of superfluous fat we may supplement remedies which tend to stimulate and sustain the heart in its natural function. These will help the reduction of unnatural adipose and at the same time protect the heart against further fatty degeneration. For this purpose the remedies mentioned previously may be used with caution. Under no circumstances will the fatty heart endure extreme stimulation and we must never employ these remedies in such strength or frequency that they "whip" the heart into unusual activity. The vaso-constricting tendency of all these remedies may be properly balanced by the addition of a vaso-dilator like *nitroglycerin.* *Crategus,* in my experience, is a comparatively safe remedy in this respect, and *cactus grand.*, in the third decimal potency, may be safely used.

The homeopathic remedies which correspond to this degenerative process are not numerous. *Phosphorus,* no doubt, comes nearest, both in its pathogenesis and its symptomatology. *Arsenicum* and *arsenicum iodid* are the next remedies to be thought of, and their action in anemic states is much better. *Calcarea phos.* and *ferrum phos.* have a far reaching action which should not be overlooked. *Sepia, silicea, sulphur, cinchona* and *natrum phos.* will give results when thoroughly indicated. *Kali iodid, aurum* and *mercurius* must be studied with care when the underlying causes are suggestive of such remedies. In addition to these I believe in the blood-iron preparations which help to correct any anemic condition.

ATROPHY OF THE HEART.

This is a rare affection and is either congenital or acquired, and may be local or general. We sometimes find a general cardiac atrophy in girls of the chlorotic type with imperfect vascular development and impairment of nutrition. Local atrophy may follow a mitral stenosis in which the left ventricle is deprived of its normal amount of blood and therefore becomes smaller. The connective-tissue proliferation of myocardial degenerations, compression from tumors, pericardial accumulations and the general tissue-atrophy from organic diseases may be considered as contributing causes.

The *pathologic features* recognized are the increased size of the organ and the diminished cavities. The myocardium is soft and anemic, while the pericardium has an abnormal amount of fat. Except the gradually increasing cardiac enfeeblement and indications of marked nutritional impairment the symptoms of this condition are negative, as are the physical signs unless we may surely outline the cardiac dulness. The *prognosis* does not offer encouragement; *treatment* gives no hope inasmuch as this is a morbid condition beyond the pale of treatment.

CARDIAC ANEURYSM.

The disease may occur as myocardial aneurysm in which the heart walls give way, or it may involve the valves alone. Aneurysm of the walls follows a chronic myocarditis of the left ventricle, which is the most vulnerable point on this account. Endocardial and pericardial inflammation may also predispose. The size of the aneurysm varies; it may be quite small or again it may attain the size of the heart itself. It occurs in the saccular form when communicating with the chamber by a comparatively small opening, or we may find the walls of the ventricle equally dilated. Concentric layers of fibrin are found on the interior of this dilatation, showing a physiologic effort toward occlusion and obliteration. Blood clots are sometimes found within and a fibroid overgrowth in the surrounding structures develop as a sort of compensatory hypertrophy.

The *diagnosis* is not easily made, inasmuch as the symptoms and physical findings are not positively differentiated from dilatation or chronic myocarditis. A pulsating prominence is sometimes found in the apex region and the area of dulness may be increased in a circumscribed manner, but when the left ventricle is equally enlarged, it simulates hypertrophy or dilatation. The *termination* of aneurysm is sudden death, unless gradual cardiac exhaustion ensues.

Valvular aneurysms can not be diagnosticated except post mortem. These develop in connection with ulcerative endocarditis, which destroys a portion of the valves and permits a globular projection in the direction of the greatest blood pressure. When the aortic valves are affected, the projection is toward the left ventricle, but when the mitral valves are affected, it is toward the left auricle. Valvular incompetency is the natural result and these signs confuse the disease with a regular valvular lesion. The prognosis is unfavorable and treatment is unavailing.

RUPTURE OF THE HEART.

This is a rare and serious accident which affects the heart. Complete rupture is exceedingly uncommon. Partial rupture occurs when the trabeculae ventriculi are lacerated so that the chordae tendinae do not hold the valves securely. Valvular incompetency follows this lesion.

The predisposing cause is found most frequently in diseases of the coronary arteries where infective thrombi or emboli lead to anemic necrosis and abscess. Myocarditis and fatty degeneration are also predisposing factors. The exciting cause is generally located in some cardiac excitement or severe muscular strain. Age also contributes to this possibility. The symptoms are not suggestive and generally sudden death is the result. Agonizing precordial pain, the signs of shock and internal hemorrhage and syncope may precede the demise. The physical signs resemble those of pericardial effusion.

The *prognosis* is of course, decidedly unfavorable and *treatment* is almost hopeless. Absolute quiet and the administration of morphin hypodermatically is about all that can be done to tide over the shock, with the hope that some measures may be applied for producing a chronic state.

CARDIAC NEUROSES.

Under this head are included all that class of cardiac perversions the cause of which is found in some disturbance of the nervous mechanism. The term functional has also been employed, as it applies to those diseases ot the heart in which no structural change exists. The typical features are noted in the perversions of the normal rhythm and character of the heart's action. These same conditions may attend organic diseases of the heart, but in the latter evidence of a lesion is present.

PALPITATION.

This relates to a violent and rapid action of the heart in which there is generally rhythmic disturbance, with some degree of precordial distress and dyspnea.

Etiology.—Palpitation may occur in connection with organic diseases of the heart, but we do not ordinarily regard this as a disease entity. Properly speaking, it is an expression of some nervous influence in which emotion and mental excitement play a prominent part. Either the inhibitory action of the vagus is abolished, or irritation of the accelerator nerves causes the heart to beat with abnormal force and rapidity. To the patient in this state of nervous tension, it seems to be more serious than it really is. Women suffer more at the period of puberty, at the menopause or when pelvic disorders cause reflex disturbances. The delicate nervous system of the female is naturally conducive to this condition. Men suffer more in later life as the result of overwork, worry or excesses. The excessive use of alcohol, tobacco, tea and coffee is often of etiologic importance. Anemia and infectious diseases, by the irritant and toxic quality of the blood, excite the cardiac accelerating nerves until palpitation is a natural reaction. In the same way we may look to dietetic and digestive errors as exciting causes. To some it is an idiosyncrasy if not an inheritance. Diseases of the central nervous system, cerebral lesions, hysteria and neurasthenia furnish a good share of cases. Environment, bad habits, reflex causes and the use of various drugs must also be regarded as factors. In fact, any cause which leads to excitement of the nervous system is a predisposing agent.

Symptoms.—The patient is often very anxious about his condition, complains of a feeling as if the heart were leaping out of its place. This often causes a choking sensation or a pseudo-dyspnea. The heart throbs violently and the pulse varies from one hundred to one hundred and fifty beats per minute. The peripheral arteries pulsate, the face is flushed, nausea, slight precordial pain and copious urination may ensue in rapid succession. All of these symptoms occur in paroxysms which generally last for an hour or longer. The seizures usually decline as suddenly as they come on. In exceptional cases this suffering may continue intermittently for days. When lesions of the central nervous system are the exciting factors, this is more liable to occur.

The *physical findings* are shown in the diffuse and forcible impulse; the same throbbing is seen in the superficial vessels. The pulse is strong, full and rapid. Percussion reveals an increased area of dulness if the overaction has caused cardiac hypertrophy. Auscultation will detect little that is abnormal except the exaggeration of the normal sound. Murmurs due to anemia may be heard at the base.

Diagnosis.—The absence of organic diseases, the presence of the causative nervous factors and the totality of symptoms as outlined above will be sufficient to make a diagnosis. The tachycardia of exophthalmic goitre must be differentiated by its persistence and the presence of the other cardinal signs, exophthalmos and goitre. The palpitation associated with valvular diseases will be recognized by their distinctive murmurs and the lack of paroxysmal symptoms.

Prognosis.—A cure in these cases should be readily obtained by systematic treatment and the removal of the exciting cause, which is not generally organic. Violent and persistent paroxysms may induce cardiac hypertrophy. Unless some degenerative process prevails, there is no danger of death from this cause alone.

Treatment.—The paroxysm must be controlled by the relief from all surrounding excitement. The patient should be put to bed in a quiet room and away from all disturbances. *Gentle massage* or a hot bath may act as sufficient sedatives, though a hot application or sometimes an icebag applied over the heart are often necessary to subdue the forcible throbbing. *Stimulating drinks* may give temporary aid, but they are liable to encourage unpleasant reactions. In *hysterical attacks* it is best to give at the very beginning of an attack a sedative strong enough to induce speedy sleep. Too small a dose only adds a narcotic excitement. Some advise morphin or codein (gr. 1/4) hypodermatically, but I do not believe these agents are demanded unless pain is a factor. *Chloralamid* (grs. 15-25) followed by a draught of cool water, is the best relief to the excited nerves. *Trional* (grs. 10-15), *chloretone* (grs. 10-15), *chloral hydrate* (grs. 10-20) or *sulphonal* (grs. 10-20) are also good agents for this purpose. *Bromid of soda* (grs. 10-20) repeated until the patient shows signs of sleepiness, may be given at two hour intervals. *Hyoscyamus tincture* (m. 5-10), or *hydrobromate of hyoscin* (gr. 1/100) will give excellent results. *Camphor monobromated* (gr. 1) may be given for a few doses, one-half hour apart, until relief is obtained. A combined tablet containing bromid of ammonia and soda (each grs. 2), and hyoscyamus (m. 5) will gradually relax the nervous system when given at one-half hour intervals for three or four doses. *Passiflora* (m. 30-60) will often subdue the nervous excitement, if it does not induce sleep. The elixir of *ammonium valerianate* in teaspoonful doses, two or three hours apart for a few doses, will give a most acceptable relief to the nervous tension and precordial excitement. In this connection we may refer to the homeopathic preparation of *valerianate of ammonia* which, in the second decimal potency, may be administered every half hour, four grains at a time, until the desired action is obtained. This is one of our best expedients when absolute enforcement of sleep is not promptly needed.

In the *interparoxysmal stage* it is necessary to give most careful attention to the regulation of the diet and other causes which tend to this disturbance. The rest cure has been strongly advocated and no doubt it is efficacious in certain instances, but the dangers of overfeeding must be borne in mind. The nervous features should be studied and treated in accordance with suggestions given under the subject of neurasthenia.

There is no excuse, except in great emergency, for treating this disease in any manner other than in accordance with its symptomatology. *Aconite* is our most valuable homeopathic remedy for the paroxysmal state. The chest constriction, the palpitation, the anxiety, restlessness and the circulatory disturbances are analogous to the symptoms of the disease. If the remedy is given in the third decimal potency at frequent intervals, it will almost invariably give more relief than is obtained by sedatives. With this remedy we should compare *arsenicum, chamomilla, coffea, cimicifuga, cactus* and *convallaria. Digitalis,* in the third decimal potency, usually gives better results in these cases than when administered in the tincture. *Pulsatilla, nux vomica* and *ipecac* will take care of the symptoms which result from gastric or intestinal disturbances. The disorders of the central nervous system are met by such remedies as *gelsemium,* which overcomes the spasmodic discharge of nervous energy, and *kali phos., zinc picrate, ferrum phos.* and *strychnia phos.,* etc., which supply the lost protoplasm of nerve cells. *Enantha, cicuta, conium, cuprum* and *ignatia* should overcome the hysteric seizures; *anacardium* covers many of the precordial symptoms when the mental excitement is aggravated. *Hyoscyamus, belladonna* and *stramonium* will also be of great service in allaying the nervous excitement which causes cardiac acceleration. In like manner we might further pursue this study of indicated remedies with satisfaction. We must be free at all times, however, to resort to expedients for temporary relief when they are demanded.

TACHYCARDIA.

This is essentially a disease of the nervous system and is well defined by the expression "heart hurry." It is a rapid action of the heart, which is generally observed in paroxysms of variable duration. We notice it in connection with nervous diseases like exophthalmic goitre, in which the sympathetic nerves are over-stimulated, while the vagus is to a certain extent paretic. It is in no sense related to cardiac lesions and it is differentiated from palpitation by the fact that it shows no cardiac sensations. Though it has paroxysmal aggravations, it may be a constant phenomenon.

Etiology.—In some individuals tachycardia is a physiologic idiosyncrasy. Again it may occur as the result of extreme excitement in neurotic patients. In certain instances the heart's action is accelerated as the result of excessive exercise, emotion, fright, grief or unnatural nervous strain. The young are liable to this, and some may increase the pulse rate at their own volition. Alcohol, tobacco, sexual excess and all the misfortunes of high life are prone to develop this irritability of the heart's action. Exhausting diseases and acute infections are often etiological factors and a paroxysmal hurry may be a sequence of almost any disease. The most typical relation is found in Graves' disease, where tachycardia is one of the cardinal symptoms which attends the progressive disturbance of the sympathetic nervous system. Other neurotic causes are found in neurasthenia, hysteria, anemia and chlorosis. Central lesions of the cerebrospinal nervous system develop a certain share of cases. Thus enlarged lymphatic glands, tumors, hemorrhagic clots and aneurysms, by their pressure upon, and degenerative process in the nerve structure, pervert the normal innervations of the brain and spinal cord, irritate the sympathetic cardiac branches or destroy the vagus control, until tachycardia is a natural sequence. Tabes dorsalis is a good example of such influences. Organic heart diseases and reflex disturbances coming from the abdominal and pelvic organs are often found to be the primary source of excitement. Environment, heredity and habit are all concerned in the production of this morbid phenomenon. In every case there is probably a composite cause arising from various physical disturbances which lead to this functional cardiac neurosis. Age and sex apparently have little influence, though it is more prone to appear in the early and middle part of life.

Pathology.—Fatty and fibrous degenerations have been found at a few necropsies, but these findings are not universally substantiated. Most of the pathologic reports rest upon theories. The early idea that a vagus paralysis was the exciting cause is not tenable in all cases. That the accelerative sympathetic action is an influence, we can not deny, but in all probability both of these factors must be taken into consideration. A bulbo-spinal neurosis, the epileptic theory and other speculations must be dismissed as without sufficient foundation. The disease with its functional characteristics has so far baffled the pathologists.

Symptoms.—While the continuous rapid heart action is a frequent expression of the disease, we must regard the paroxysmal form as more typical. This is recognized by the *sudden onset,* followed by a rapid heart action, which may reach 180 or more beats per minute. The *pulse* is small, weak and occasionally irregular. The pulse waves may be so rapid that they run together and are uncountable. Auscultation is, therefore, necessary for a true record of the heart's action. *Respiration* may be rapid, difficult or quite normal. A *flushed* face, headache, vertigo, throbbing arteries and a semblance of anginal pain characterizes some cases. The patient invariably complains of the rapid heart action. Tendencies to *faintness,* anxiety and restlessness are often experienced. *Hysterical symptoms* may prevail when females are afflicted. The attack usually *subsides suddenly* after a few hours and the patient is left in a state of nervous exhaustion and apprehension. In chronic cases the duration of the attack may be extended. When reflex causes are at fault, a recurrence is not so probable as when it is associated with myocardial changes or severe nervous causes. When a cardiac inadequacy permits an overdistention of its chambers, circulatory engorgement will be manifested by cyanosis, jugular pulsation and evidences of pulmonary edema.

Physical Signs.—The physical signs will show a diffuse, rapid and sometimes irregular impulse upon inspection and palpation, but percussion does not reveal the dulness of hypertrophy, unless in long standing cases. Auscultation detects an accentuation of the first and a weakness of the second aortic sound. The pulmonic sound may be accentuated. An apical systolic murmur may be heard, while the carotids pulsate and sometimes reveal a murmur. During the interparoxysmal stage murmurs are not found unless there is a valvular lesion. A sphygmographic tracing shows a rapid cardiac action, with low arterial tension, and this is considered a valuable sign.

Diagnosis.—The foregoing symptoms, which consider both cause and results, ought to enable one to make a diagnosis. *Palpitation* does not have so high a pulse rate and it has, in addition, the symptoms of dyspnea, precordial constriction, a smothering sensation and nervous anxiety, attended with pain.

Prognosis.—While there is no particular danger to life, the hypertrophic results may favor cardiac impairment, particularly in old people. Much depends upon the causative lesion and the resisting power of the cardiac muscle. When there is an organic cause involving the central nervous system, the outcome is not hopeful. The symptomatic use of remedies will afford a great deal of relief, but a cure will depend upon a removal of the underlying cause.

Treatment.—The treatment will be somewhat similar to that of cardiac palpitation, but it will vary in accordance with the cause. The use of remedies on the symptomatic principle will yield the best results. Rest, diet and local treatment should be the same as described under palpitation. The neurotic phase and much pertaining to the remedial features are described best under the subject of exophthalmic goitre. The use of the cardiac tonics may be demanded in some cases, but the best results will come from the homeopathic remedies, which, relieving the nerve irritation, safely overcome the arterial tension. Thus *aconite* and *veratrum viride* may be used with persistency from the very beginning. The nervous excitement is easily quieted by *valerianate of ammonia, coffea, chamomilla, ignata, agaricin* and *hyoscyamus,* while the deeper lesions respond to the continuous use of indicated remedies like *zinc phos., picric acid, aurum chlorid, arsenicum iodid, potassium iodid, argentum nitricum.*

The paroxysm of the hysterical state is greatly relieved by the internal use of *moschus, ignatia* and *nux moschata. Cinchona, china arsenicum* and *nux vomica* will, together with the *ferrums,* be thought of when anemic and digestive disturbances are factors. *Caffein valerianate, asafetida, cimicifuga* and *camphor* are often similarly indicated when nervous symptoms predominate. *Gelsemium, spigelia, scutellaria* and *sepia* will demand consideration in some cases.

BRADYCARDIA.

Bradycardia or brachycardia is the opposite of tachycardia and refers to a slow action of the heart. Under this condition we find the pulse rate decreased to sixty beats or less per minute.

Etiology.—It is associated with neuroses like epilepsy, hysteria, melancholia, mania, etc., but it is more particularly recognized as secondary to other diseases. It occurs frequently during the convalescence from acute infectious diseases. We often find a pulse rate ranging between forty and fifty beats following a case of pneumonia, typhoid fever, influenza, diphtheria, rheumatic fever and other infections. It often arises from gastro-intestinal or hepatic diseases when auto-infection is a factor. The toxins of uremia, diabetes and catarrhal jaundice, and drugs such as digitalis, alcohol, aconite, tea and coffee are also cardiac depressants. Anemia, chlorosis, gout, fibroid and fatty myocardial changes, emphysema and asthma, and many of the chronic diseases predispose to a retarded action of the heart. Old age often displays a pulse rate ten to fifteen beats below the normal. Degenerative and other lesions of the medulla may cause it when the vagus center is affected, thus displaying a gradual slowing of the heart's action by a stimulation of that nerve.

Symptoms.—The retarded pulse is the chief characteristic. This is a permanent condition in some cases, but is more liable to exist as paroxysmal, appearing suddenly and terminating in a similar manner. The pulse is weak and small in character and the patient may suffer from extreme physical exhaustion. When there are complicating valvular lesions, the force of the cardiac action may be increased, but this does not increase its frequency. The cardiac impulse and sounds are feeble. Auscultation will be necessary to obtain the relation between the heart action and the peripheral pulse, as the heart may be unable to transmit every beat. Sometimes there are prodromal symptoms such as tinnitus, vertigo and nervous excitement preceding the paroxysm. *Stokes-Adams Syndrome* is a term used to define an attack of bradycardia attended by unconsciousness of short duration, disturbed respiration, and occasional convulsions. This may also occur in other heart diseases in which the vagus and medulla are involved.

Diagnosis and Prognosis—A pulse under sixty per minute, appearing paroxysmally, is sufficient for a diagnosis. The heart's systole should show a corresponding slowness and usually we shall be able to find the predisposing causes. With any functional cause like anemia and gastric disorders, the prognosis will be comparatively favorable. The neurotic causes should be looked upon as of grave import. Sudden death is liable to occur.

Treatment.—Stimulation seems to be an absolute necessity in this disease. The various cardiac tonics are usually indicated, though they must be given in small doses. *Strychnia, caffein* and *nitroglycerin* are demanded in the serious paroxysms. *Whiskey* and *ammonia* may be used in some cases. *Adonis vernalis, apocynum, cannabis indica, kola* and *strophanthus* have been recommended for this condition.

CARDIAC ARRHYTHMIA.

Arrhythmia, as the term implies, is an irregularity of the heart's action and pulse. It occurs as a complication of organic or functional diseases and in some cases it may be simply an idiosyncrasy. A neurosis is frequently a fundamental cause. The character of this irregularity may affect the volume or the time of the heart beat or pulse beat. Several varieties of this arrhythmical action are recognized. (1) *Intermittent* is the term applied when there is an intermission or dropping of the cardiac beat. This may appear at irregular intervals, or it may occur with cyclical regularity at every second, fourth, sixth or eighth beat. It is defined as *deficient* when the systole is omitted. The systolic action, in some cases, may be so weak that it does not produce a perceptible wrist pulse, though there is a feeble first sound on auscultation. (2) *The bigeminal* and *trigeminal* pulse represent a pulsation of two or three beats in rapid succession, followed by a marked interval. In cardiac bigeminism the second systole is so weak that it does not always reach the radial artery and hence we may detect only a single pulse beat. (3) *Embryocardia* or *fetal rhythm* simulates the action of a fetal heart in which the long pause is shortened and the character of the two sounds is identical. We notice this in the later stages of fever and in extreme dilatation. (4) *Pulsus paradoxus* represents a condition in which the beats are more frequent and less forcible during inspiration. It is associated with pericarditis during the stage of effusion and in other pathological conditions which weaken the heart. It is presumed to be at times a physiologic condition, as it may be observed in sleeping children. (5) *Alternate irregularity* is recognized when weak and strong contractions alternate in the pulse beat. (6) *Delirium cordis* applies to a condition of extreme irregularity, with a variety of rhythmical disturbances. (7) *Gallop rhythm* is a term used when the sounds resemble the hoof-beat of a galloping horse. It is defined by the expression rat-tat-tat; the first, or, more commonly, the second sound, seems to be split and it indicates weakness of the heart muscle. It is a serious sign in connection with nephritis. Anemia of long standing, arteriosclerosis, dilatation and the myocardial degenerations following acute infections contribute to its occurrence.

Etiology.—Baumgarten's Classification is quoted in Osler as follows: "(1) Those due to central-cerebral causes, either organic disease, as in hemorrhage, or concussion; more commonly psychical influences. (2) Reflex influences, such as produce the cardiac irregularity in dyspepsia and diseases of the liver, lungs and kidneys. (3) Toxic influences. Tobacco, coffee and tea are common causes of arrhythmia. Various drugs, such as digitalis, belladonna and aconite, may also induce it. (4) Changes in the heart itself. (a) In the cardiac ganglia. Fatty, pigmentary and sclerotic changes have been described in cases of this sort and may have an important influence in producing disturbances in the rhythm; but as yet we do not know their exact significance. They may be present in cases which have not presented arrhythmia. (b) Mural changes are common in conditions of this kind. Simple dilatation, fatty degeneration, and sclerosis are most commonly present, the latter two usually associated with sclerosis of the coronary arteries."

Symptoms.—The typical signs of arrhythmia are often overlooked in functional cases and it is only in association with serious complicating diseases that attention is called to the heart disturbance. The physical signs are not distinctive except as organic changes direct attention to some pathologic perversion.

Diagnosis.—To determine the condition of arrhythmia it will be necessary to make a careful and frequent examination of the pulse and the heart at stated intervals. The sphygmograph is the best means for the detection of the irregularity. The functional and organic causes are also differentiated by a painstaking investigation in accordance with diagnostic methods heretofore mentioned.

Prognosis.—Arrhythmia does not endanger life when it is of functional character, or when a reflex cause will yield to treatment and proper care. Valvular and myocardial causes are, of course, grave, but much depends upon the early diagnosis and the careful management of the case.

Treatment.—The milder forms of this disease will respond to the use of the indicated remedy. The valvular diseases demand cardiac stimulants, while rest and freedom from excitement will give much relief in the average case. The treatment in general pertains to the primary disease, and this feature has been covered under different chapters. The high arterial tension is modified by the use of *nitroglycerin,* but when dilation is threatened, *digitalis, strophanthus, adonis vernalis, convallaria* or *crategus* will give better results. The reflex influences must be corrected in accordance with treatment suggested for these predisposing diseases. Neurotic causes will respond to treatment described under neurasthenia.

ANGINA PECTORIS.

Angina pectoris, or stenocardia, is a paroxysm of precordial pain which radiates along the larger cardiac vessels and is accompanied by a feeling of constriction of the chest. The nervous symptoms are extreme and the patient has the fear of impending death. It is symptomatic of various cardiac diseases and particularly those in which morbid changes develop in the root of the aorta and in the coronary arteries.

Etiology and Pathology.—The morbid anatomy and the etiology of angina are so intimately associated that it seems best to discuss them under the same head. So far as the history of the disease is concerned, a volume might be written to cover the various theories of different authors. It was once thought to be a condition of cardiac spasm in which the muscular balance was at fault. The neurotic inheritance and the rheumatic, gouty, syphilitic diathesis and other causes have always been considered, and they possibly still have some bearing upon the subject. A vasomotor form has been described in which the pain is charged to the tension of the ventricular walls from the general vascular spasm. The popular idea at present is that it is due to a neuritis affecting the cardiac plexus of nerves and the radiating pain is explained by the distribution of these nerves along the coronaries and the aortic arch. The relation of this plexus to the vagus and sympathetic system will also account for the dissemination of pain and the characteristic symptom of constriction. The cause for this neuritis must be found in the pathology which predisposes to a sufficient irritation. While various lesions have been revealed post mortem, it must be emphasized that the essential cause is an interference with the blood supply of the heart muscle. Coronary sclerosis is looked upon as the most frequent pathologic factor. General arteriosclerosis, aneurysm and associate atheroma, valvular diseases (especially aortic) and lesions of the aorta may be mentioned as other pathologic possibilities.

When *coronary sclerosis* is the cause, we shall find that the mouths of the coronary arteries are narrowed by the sclerotic process leading to ischemia of the heart muscle. Pain of the anginal character arises as soon as any undue exertion is made or a sudden emotion creates unusual heart action. Then it is impossible for the narrowed coronaries to supply the heart with a sufficient amount of blood and the ischemia is so intensified that the nerve filaments are irritated and the anginal attack follows. Aneurysm and atheroma of the ascending aorta may lead to a similar narrowing of the coronaries and a consequent anemia of the heart muscle. Aortic regurgitation decreases the blood pressure so that cardiac ischemia is a natural result and this may be said of mitral and aortic stenosis, which oppose the entrance of a normal amount of blood into the cardiac substance. Thus it may be figured out that almost any cardiac lesion is liable to develop a muscular starvation sufficient to predispose to anginal attacks. Myocardial changes are favorable to a fatal termination during the paroxysm.

The *excessive cardiac irritation* which is secondary to this enforced anemia initiates the paroxysm. Hurchard claims that "the heart itself is the starting point of the attack. From here the stimulus ascends by way of the sensory centers and finally reaches the medulla. Thence it is reflected along the intercostal nerves and bronchial plexus as a manifestation of pain. The stimulus next reaches the vagus center and from here and inhibitory impulse is sent down to the heart, the original starting place, and declares itself by slowed and possibly an intermittent action of the heart." This leads Babcock to infer that "the inhibitory action accounts for the sense of constriction and the fear of death, as well as the occasional dilatation of the cardiac cavities." He further states that "in pseudo-angina, on the contrary, the point of departure of the irritation is an intercostal nerve or some other peripheral or visceral nerve, whence, as in true angina, it also passes to the medulla. From there an impulse is sent out, not along intercostal nerves and the brachial plexus, but is passed down along the vagus or accelerator nerves of the heart to this organ. According to the route thus selected, the action of the heart becomes either rapid or slow and otherwise disordered." The above quotation is made verbatim because it offers a clear understanding of the differentiation of true and pseudo-angina.

Age is a powerful factor which predisposes to angina pectoris of coronary origin. After the fortieth year arteriosclerosis is liable to exist and thus angina occurs more frequently in people beyond middle life. Males are affected mostly and probably as a result of high living. The exciting causes come from the sudden raising of the blood pressure in the aortic circulation or the condition of cardiac ischemia, which demands more work of the heart than it can perform with its diminished blood supply. Extreme muscular effort is the most common cause which suddenly raises the blood pressure. Emotion, anger or any excitement which accelerates the heart, when the blood supply is reduced, are exciting factors.

Symptoms.—The attacks come on generally during some unnatural excitement or exertion and the typical symptoms are not always observed by the physician. Death is liable to be a sudden termination. Prodromal symptoms are uncommon. The paroxysm *begins suddenly* and the patient complains of a *severe pain* and a *sense of constriction* in the precordium. The pain may affect the whole chest, causing almost a general convulsion. It radiates most frequently to the left shoulder, then to the back, neck and down the arms to the fingers. It may extend to the abdomen or genitals, the focus of greatest intensity changing during the paroxysm. Usually, however, the pain is most intense in the chest region. During exertion the patient may be observed to stop suddenly, grasp for something and put the hand to the chest. The *countenance* is pale and suggests terrible anguish. *Suffocation* is extreme and the body is bathed in a clammy sweat. The *respirations* are shallow or temporarily arrested. The sense of impending death gives rise to constant fear. The *pulse* shows high tension in the beginning of a paroxysm, but it tends toward the subnormal character as the circulatory failure increases. The rhythm of the pulse may not be disturbed. In fact, it varies in accordance with any organic cardiac condition which may coexist. The *paroxysms* last from a few seconds to two or three minutes, though in rare cases they may continue longer. A recurrence is liable to take place whenever the proper exciting causes act. As relief is experienced vomiting, eructation of gases or the passage of large quantities of urine takes place. The *severity of the attack* depends upon the pathologic cause and the cardiac condition. It seems plausible to accept the term pseudo-angina when the cause is found alone in a neuropathic state, hysteria or general anemia. The differentiation of such conditions will be taken up under diagnostic consideration.

Diagnosis.—The sudden and intense pain in the substernal and parasternal regions, together with the pain and the chest constriction defined in the symptomatology, should point unmistakably to angina. The short duration and the sudden cessation of the cardinal symptoms will help in making the diagnosis, providing we find the other pathologic and etiologic features which develop the symptoms. *Pseudo-angina* starts with a history of hysteria, neurasthenia, toxemia or reflex disturbances. It occurs often with women and at any age. The paroxysms are inclined to be periodic and of longer duration. The pain is not so severe and is diffused over the chest. The nervous excitement and emotional features are more pronounced and the arterial tension is not increased.

Prognosis.—*Sudden death* is always a possbility in anginal attacks and the pathology is such that a cure is not to be expected. Patients may survive several paroxysms, while some cases succumb to the first attack. Appropriate treatment and the most exacting care as to diet and habits may delay the end for many years. Abnormal muscular strain and nervous excitement should be guarded against.

Treatment.—Violent exercise and emotional excitement must be constantly restricted. The general health must be carefully watched and any contributing cardiac disease must be relieved as much as possible. It is highly essential to encourage the nutrition, which necessitates a careful study of dietetic principles. This is for the purpose of preventing the recurrence of paroxysms. The *treatment of the attack* demands the most careful management of the patient to allay the fear and control the mental excitement which enhance the danger. Prompt relief is the essential of importance. For this purpose no better agent is known than the inhalation of *amyl nitrite.* This is so conveniently made in the form of perles that the patient may always have one at hand. *Nitroglycerin* and *nitrite of sodium* have been recommended highly, but they do not give such immediate relief to the features of spasm. *Erythrol tetranitrate* for prolonged use may give better results. *Chloroform* and *ether* inhalations and *aromatic ammonia* (m. 10-15) are useful in many cases. *Opium* is regarded as one of the best remedies. As its effect by mouth is not rapid, morphin hypodermatically administered is a better agent. Enough of this should be given to subdue the pain and all nervous excitement. It not only overcomes the muscular spasticity and pain, but its action as a sedative and cardiac tonic is unsurpassed at a time like this. Some physicians prefer a tablet containing one minim of a 1 per cent solution of *nitroglycerin,* repeated every two to five minutes for three doses if necessary.

In the milder cases *aromatic ammonia, whiskey* or the *elixir of valerianate of ammonia* may suffice. These remedies however, should not be relied upon in the pronounced cases. Pseudo-angina will yield to the management and the remedies usually employed in the treatment of hysteria and neurasthenia. During the paroxysm we must not employ aconite, veratrum, strychnia or digitalis. Hot flannels saturated in alcohol and water will often be of adjuvant use, and the icebag, applied to the precordium, is frequently efficacious in warding off an attack.

During the *interparoxysmal state* we should resort to remedies which will subdue the nervous excitement, and at the same time have a tendency to gradually lower the blood pressure. For this purpose a diet free from meat should be advised. Among the remedies we should mention *arsenicum iodid* first. This should be given for a long time in the third decimal potency for the sake of its gradual tonic action. *Potassium iodid* or, better, *strontium iodid,* may be considered for the reparative action for which these drugs are generally used. *Spigelia, bryonia, cuprum, cimicifuga, kalmia* and *lachesis* are remedies to be considered in accordance with their symptoms. *Lilium tigrinum, cactus, naja* and similar remedies may be employed whenever they are indicated. The Nauheim baths and the resistance exercises will help to restore the cardiac health.

CONGENITAL AFFECTIONS OF THE HEART.

Cardiac structural defects are present at birth as the result of fetal endocarditis, or are due to arrested development. Fetal endocarditis is a rare affection. The pathologic changes are found most frequently on the right side of the heart inasmuch as this sustains the greater strain during fetal life. The abnormalities may be discovered at the pulmonic, aortic or auriculo-ventricular orifices. The morbid feature partakes of the chronic sclerotic character. The endocarditis leaves the segments in a smooth, thickened and contracted condition. The cusps may be united and the chordae tendineae are frequently thickened and shortened. Nodular bodies on the mitral and tricuspid segments, which are normal to fetal life, may remain after birth, but these must not be confounded with the pathologic changes of fetal endocarditis. They are known as the nodules of Albini. Smaller bodies, the result of minute antenatal hemorrhages, may also persist after birth.

Stenosis of the pulmonary orifice is the most common congenital affection. A slowly developing endocarditis causes an agglutination of the segments until the orifice is greatly narrowed. Stenosis of the conus arteriosus may coexist with this. Atresia, or obliteration of the pulmonary orifice is a less frequent malformation. The aortic orifice is not often involved. Tricuspid stenosis is slightly more frequent, while mitral disease is rare.

The *cardiac anomalies* related to arrested development present more varied features. Of the general defects of development we find many types. *Double heart* is associated with other deformities. *Bifid apex* is an occasional condition. *Ectopia cordis* is the result of imperfect development of the chest wall.

The heart may be found in the cervical, pectoral or abdominal regions. A stillbirth is generally the history of these cases. *Dextrocardia* is defined when the heart is found in the right side of the chest, the descending aorta being displaced to the right of the spinal column and the arch is reversed. *Mesocardia* is a term applied when the heart occupies the median line, thus preserving the normal fetal position. *Imperfect closure of the foramen ovale* may be an independent malformation, and is also often associated with obstruction of the pulmonary orifice.

Developmental defects affecting the whole or a part of the auricular or ventricular septa may exist. These irregularities may reduce the heart to two or three chambers. Rudimentary increase or decrease in the number of segments of either the aortic or pulmonary valves are occasionally found. This is due both to defective development and to cohesion following endocarditis.

Symptoms.—*Cyanosis* is the most distinctive symptom, and it is usually noticed immediately after birth. The discoloration is most noticeable about the lips, mucous membrane of the mouth, nostrils, fingertips and the ears. From the extremities it may extend to the whole body. It may not be observed always at birth, but becomes manifest shortly after when the child overtaxes the heart by a crying spell. *Acute pulmonary inflammations* also augment the symptoms. Should the child live, the fingers are clubbed and the nails are thickened and curved. The skin is blue, the pulse small and weak, the temperature is subnormal, dyspnea and cough appear from the slightest exertion and the other concomitant symptoms of cardiac disturbance are usually present. Convulsions, cerebral abscess and pulmonary affections are possible sequelae.

Diagnosis.—To confirm the diagnosis we shall look for the early history of a persistent cyanosis, slight enlargement of the right ventricle, with but little extension in the area of dulness as the child grows older. The cardiac impulse is feeble and a systolic murmur—which may become loud or musical—is heard near the pulmonic orifice. This has a small area of transmission upward and to the left. The mental faculties do not show a normal development and the body's growth is stunted. Other valvular defects may develop if the child reaches the age of puberty.

Prognosis.—The prognosis must always be looked upon as exceedingly grave. Death usually occurs shortly after birth.

A small number live a few years. Pulmonary stenosis gives more hope for existence, if not for a healthy life.

Treatment.—Little can be said for the treatment, except the hygienic care which must be considerately given from the very first. Marked attention is necessary in order that the child be kept very warm all the time. If life is spared and there is any prospect of remedial relief for this congenital defect, it will be obtained by means of the usually indicated remedies. A constant care must safeguard the growth of the child.

DISEASES OF THE BLOOD VESSELS.

ARTERIOSCLEROSIS.

Diseases which affect the arteries are not numerous and have been studied under various names expressive of the cause and the pathologic condition. The inflammatory and degenerative processes are both concerned in the descriptive illustrations. For instance, the chronic inflammations have been included under the terms *endarteritis obliterans,* in which the arterial lumen is reduced or obliterated, and *endarteritis deformans,* which refers to an increase of the vessel's calibre. The fatty, lardaceous, calcareous, tuberculous and any other degenerations which affect other parts may pertain to the arteries. *Arteriosclerosis* is the most common form, and represents a connective-tissue overgrowth in the arterial coats which is followed by calcareous depositions. The intima shows the most extensive changes.

Etiology.—*Advancing years* are the most natural predispositions of this disease. Even when unattended by positive morbid signs, age tends to lessen the normal elasticity of the arterial wall. In order to compensate for the dilation of the vessels which follows this loss of elasticity the intima thickens proportionately. It is a possible occurrence in those of middle age and even in younger subjects, while *heredity,* no doubt, has something to do with its etiology. *Syphilis* is the most common toxic cause, and *alcoholism, lead poisoning, gout* and *chronic nephritis* are also inciting factors in the degenerative process. *Occupations* which induce muscular strain or constant physical exertion which persistently increases the blood pressure, may be the only discoverable source. And this is the explanation why it is more prevalent with the male sex. *High arterial tension* favors it in that class of people who indulge in the excess of eating and drinking. On the other hand, a diet which impoverishes the system may cause a systemic impairment which leads to arterial degeneration. It occurs also in the pulmonary artery and sometimes in the pulmonary veins when there is, from any cause, a prolonged high pressure in the lesser circuit. When there is *hepatic cirrhosis* the portal vein may suffer a similar sclerosis. The splenic, hepatic and superior mesenteric arteries are also affected in corpulent persons of sedentary life. *Mechanical obstruction* and the subjection of an artery to undue strain and alternate relaxation are often contributing causes. An example of the latter is found in the arteries of the arms of laboring men and in the cerebral vessels of mental workers. Many other causes probably exist which upon investigation will be found to conduce to this degenerative process.

Pathology.—Arterial sclerosis most frequently affects the aorta and the coronary arteries. The cerebral, temporal, radial, brachial, ulnar, femoral and iliac arteries are also liable to this involvement. It is either circumscribed or diffuse. In the *circumscribed form* yellowish-white or gray atheromatous patches are discovered in the aorta or other arterial branches near their orifices. These areas are at first translucent and soft, but the degenerative process gradually undergoes a calcareous change. The middle and external coats are first affected and the intima follows with a secondary or compensatory thickening. This thickening of the intima is the result of connective tissue development, but when the degenerative process commences aneurysmal dilatation of the vessel is liable to take place.

In the *diffuse variety* there is a more general distribution of the morbid elements, though the circumscribed form may still be evident in the aorta which is liable to dilatation. The intima is decidedly thickened and the other coats are affected to a slighter extent. The subendothelial connective tissue proliferates, the media shows a hyaline transformation and the muscular fibers and elastic tissue have a tendency to disappear as the disease advances. Calcareous deposits may now develop until the vessel wall is greatly thickened, or the arterial structure may be transformed into a hard and unyielding mass.

Necrotic degeneration is found in the smaller arteries of advanced cases. The surfaces may also undergo ulceration until abscesses and thrombi are natural sequelae.

The *general effect* of arteriosclerosis produces aneurysmal tendencies and for the same cause there may be a lack of blood in other organs—the heart, kidneys, and liver—to the extent that the nutrition is cut off sufficiently to create atrophy. When the coronaries are affected fibrous myocarditis and sclerosis of the aortic and other segments may follow. During the progress of the disease the arterial pressure is elevated on account of the resistance to the blood current by loss of arterial elasticity. The arteries accessible to palpation will be tortuous and rigid, while the pulse is full, hard and sharp.

Symptoms.—The symptoms depend decidedly upon the location of the pathologic process. They are often so latent that they are not discovered until the disease is well advanced, or there are signs of circulatory disturbance affecting the heart muscle, the kidneys, brain or digestive organs. The extremities are usually cold. *Palpitation, dyspnea* and *precordial pain* are detected when cardiac hypertrophy follows the increased peripheral resistance. The *apex beat* in this case will be decidedly displaced downward and to the left. The first sound is weak and the aortic second sound has a marked accentuation. An apex systolic murmur is often present when myocardial degeneration exists. The *kidney disturbances* are not always found at first as these organs may suffer gradual atrophic changes following the diminished blood supply. There is usually an early increase of *pale colored urine* with low specific gravity. The urea is diminished and later albumin appears, together with a few hyaline casts. In the cerebral type of milder form, headache, tinnitus, vertigo and syncope are suspicious symptoms. In older patients a thrombus or an embolus may give rise to apoplexy and secondary cerebral softening as the result of anemia. *Digestive disturbances* do not suggest very much as they are so often in evidence from other causes. *General circulatory failure* is, however, a symptom to be explained. There are sometimes suggestions of pulmonary atheroma, and arteriosclerosis of a peripheral type is recognized when tissue starvation, due to an obliteration of an artery of the extremities, is evidenced by local gangrene.

By *palpation* it is possible in advanced cases to detect the hardened radial, temporal, brachial or femoral vessels. In some cases this is utterly impossible. The pulse is slow and hard, showing the high arterial tension. As the rigid arterial walls make palpation uncertain, Anders suggests that compression be performed with the index finger, while the middle finger feels for the pulse wave; if this is found, arteriosclerosis is probable. The sphygmographic tracing shows a gradual upstroke, a broad and flattened summit, with an absent or diminished dicrotic notch.

Diagnosis.—Hardened arteries, high-tension pulse, hypertrophy of the left ventricle and accentuation of the aortic second sound, are the characteristic symptoms upon which a diagnosis is based. The signs which point to circulatory impairment and the invasion of other organs as enumerated in the symptomatology, are also to be considered.

Prognosis.—Arteriosclerosis is a disease which furnishes many surprises. The pathology would naturally suggest the danger of sudden death from the slightest overexertion, but many patients endure the disease for years and die from other causes. As it is essentially incident to advanced age, any of the unfavorable symptoms may appear unexpectedly. Everything depends upon the degree of vascular change and the extent of visceral disturbance. Renal complications result in gradual asthenia, while cerebral hemorrhage usually ends life suddenly.

Treatment.—The protection of sclerotic arteries should be the first consideration on the part of the physician. It will be necessary to regulate the habits, diet and the occupation so that undue vascular strain may not take place. A safe system of exercise should be instituted and the occupation should not permit excessive muscular effort. A quiet outdoor life is best. Overeating and foods which fatten or excite the circulation must be avoided. A *light diet* with vegetables in excess is best. The patient should be warned to eat sparingly and regularly. Digestive disturbance must be corrected and the kidneys should not be excited by nitrogenous foods. A normal renal and hepatic activity and a healthy skin are mandatory requirements for the sake of toxin elimination. *Sleep* is another means for the preservation of circulatory quietude. This should be prescribed in the day time as well as night, for every moment of rest reduces the tension of the weakened arteries. The best thinkers in medicine are at last arriving at the point where they state that the most acceptable mode of practice must be in accordance with the symptoms or, as they are pleased to say, "symptomatic."

Regarding *remedies,* it is natural to refer to the experience of practicians as well as to the scientific theory of symptoms. It has long been a settled opinion that it is best in many cases for the temporary relief at least, to resort to the vasodilator remedies. The best of these is *nitroglycerin* (gr. 1/100-1/50), given often enough to maintain the dilatation. *Erythrol tetranitrate* (gr. 1/2 t. d.) is a new vasodilator, whose action is more prolonged than nitroglycerin. This may be combined with *digitalis* if the heart needs the sustaining force of the latter remedy. In general the vasoconstrictors must be avoided, unless they are demanded for other symptoms. If these are needed in cardiac insufficiency *strophanthus* is one of our best agents. For the relief of vascular tension it is absolutely necessary to keep the bowels free and the liver in a most active state. The older method of using calomel is not best, though it may be resorted to when an active purge seems essential. It is possible in the majority of cases to obtain this end by the use of remedies which do not produce such unfavorable results, such as *cascara, castor oil* or *chinoidin. Venesection* is a procedure which is bound to receive more consideration in the future in cases when catharsis fails to relieve the circulatory tension. Much of the treatment which pertains to cardiac lesions will apply here. It is also necessary in all cases to direct our attention to the other organs which may be involved.

The internal remedies which are still in use usually are combinations of iodin. The *iodid of sodium* (grs. 2-5), three or four times daily, has received considerable commendation. The dosage may be systematically increased. *Iodid of potassium* and *iodid of strontium* are favorite remedies with some. The *iodid of lead,* the *iodid of arsenic* and the *iodid of lime* may be considered. *Cuprum, ferrum iodid, ledum* and *zinc* are remedies which will always bear close study in these cases. *Mercurius iodid* and *aurum iodid* will naturally be thought of when arteriosclerosis is in any sense a sequela of syphilis. *Nitric acid* should not be overlooked when tertiary symptoms are present, and following this last remedy we will do well to look carefully for the indications of *calcarea carb., kali carb., natrum carb., hepar sulphur* and *graphites. Secale, lachesis* and *phosphorus* have also a definite relation to the metamorphosis of tissue which is so prominent a factor in this disease.

ANEURYSM.

The term aneurysm properly means a circumscribed dilatation of an artery. This must be differentiated from the uniform dilatation of a vessel which may follow sclerosis. The subject is studied under two heads, (1) true and (2) false aneurysm. A *true aneurysm* is recognized when the dilatation is circumscribed and defined by one or more of the arterial coats. A *false aneurysm* is a circumscribed collection of blood found in the surrounding tissues having passed out through a rupture in the arterial walls for which hematoma is a more correct term. Other terms have been applied in accordance with the forms which aneurysms take. For instance, a *dissecting aneurysm* is one in which the blood passing through a rupture in the intima burrows between the inner and middle coats or in the layers of the media, thus separating them for some distance. A *sacculated aneurysm* resembles a sac protruding from one side of the artery; the artery and tumor may communicate either through a large or a narrow opening. A *fusiform variety* is represented by an enlargement of the vessel which is quite uniform. An *arteriovenous form* is one in which a fistulous connection exists between an artery and a vein. This is called an aneurysmal varix. It may appear as a sac between the two vessels under which condition it is defined as a varicose aneurysm. *Miliary aneurysms* are found along the course of the cerebral vessels and receive the name from their minute size.

Etiology and Pathology.—Aneurysm is possible when the mural resistance is weakened and the arterial pressure is increased. *Arteriosclerosis* is the most frequent cause. Prolonged high arterial tension which attends persistent laborious work or which follows cardiac hypertrophy is a common predisposing cause. *Sudden and severe strain* or violent coughing spells often induce it. *Traumatism* may be regarded as an occasional factor and all the predisposing causes of arteriosclerosis such as syphilis, gout, alcohol, rheumatism, nephritis, etc., must be thought of in our study of the disease. *Non-septic embolism* may create an aneurysm on the proximal side of the obstruction. In *malignant endocarditis* the inflammatory invasion may extend to the aorta and there favor an aneurysmal dilatation by its degenerative process. Men are afflicted oftener than women and the predisposing age is between thirty and fifty. *Heredity* may be a factor to the extent that the arteries are naturally of poor quality. The intima and adventitia are thickened, though the media probably yields to the degenerative process first. Fibrous bands supplant the muscular tissue and the walls gradually become thin until a rupture takes place. The blood in the tumor is partly fluid and contains clots. Coagulation about the circumference tends to obliterate the tumor to a greater or less degree and seems to be nature's effort to strengthen the thinned and dilated wall. Through dislodgment of these thrombi, embolism of a distant vessel is a constant danger. Pressure upon neighboring tissues and organs and ruptures constitute the greatest dangers. The reacting influence upon the heart is an eventual menace. According to their location the following varieties may be considered:

ANEURYSM OF THE THORACIC AORTA.

The aorta is the most common seat of aneurysm, and the thoracic portion is affected more than any other. Half of these cases occur in the ascending portion, while the transverse and descending parts are invaded less frequently. The causes which favor their development are found in the etiology of arteriosclerosis, and alcohol or syphilis may be discovered as primary factors. When arterial degeneration exists, the additional strain of physical exertion may be sufficient to lacerate the media and distend the arterial coverings until the aneurysmal pouch is found. The size and shape of the aneurysm varies decidedly. The majority are saccular and some may attain the size of a cocoanut while smaller ones often develop near the root of the aorta. The larger sacs may invade the upper thorax and cause considerable trouble by compression upon the trachea, bronchi or lungs. Instances have been found in which erosion of the vertebrae has taken place, the protrusion appearing in the back. Secondary pouches or multiple tumors sometimes characterize the formation.

Symptoms.—The symptomatology depends greatly upon the size and location of the tumor. Some cause neither symptoms nor physical signs, either on account of their latent development or because they do not seriously affect neighboring tissues. Sudden death as the result of rupture may be the first indication. *Pressure symptoms* and the presence of a pulsating tumor are the most positive signs. Aneurysms of the ascending portion of the arch, by their compression of the superior vena cava, cause engorgement of the veins of the head and arm, and if the subclavian is pressed upon, edema and enlargement of the right arm is observed. Should the inferior vena cava be affected, we should look for similar symptoms in the lower extremities. Large aneurysmal tumors displace the heart outward to the left, and in case the right recurrent laryngeal nerve is pressed upon, aphonia and dyspnea are experienced. Rupture into the pericardium, pleura, or, rarely, the superior vena cava, are possibilities to be feared. External rupture is a less common termination. If the aneurysm is exceptionally large, it may erode the ribs and sternum, causing a tumor at the second or third interspace.

Aneurysms of the transverse arch most commonly extend backward, creating symptoms of cough and dyspnea if the trachea is pressed upon; dysphagia often exists when the esophagus is compressed. The bronchus may be affected in like manner giving rise to dyspnea and such secondary conditions as bronchiectasis, bronchorrhea and lung suppuration. When the left recurrent laryngeal nerve is pressed upon, aphonia will follow. The upward extension of the sac will result in sympathetic nerve disturbance and carotid, innominate, and subclavian pressure. Compression of the thoracic duct is sometimes noticed, rapid emaciation resulting. The sac may in some cases be felt at the suprasternal notch.

When the *descending portion of the arch* is affected there will be signs of pressure on the spinal cord if erosion of the vertebrae is sufficient to permit the encroachment upon it. Dysphagia and bronchial symptoms may arise from the same compression. The cord irritation is expressed by severe and paroxysmal pain. The tumor may reach the subcutaneous tissue in the region of the left scapula. Rupture into the left bronchus, pericardium, pleura or superior vena cava ends in sudden death. The lower part of the descending aorta may be the seat of aneurysmal dilatation and, in this case, it will affect the structures nearer the diaphragm, often showing a tumor in the umbilical region.

The *general symptoms* of aneurysm will always be characterized as follows: *Pain* is an early and constant symptom. It may occur in paroxysms and varies greatly with the location and development of the tumor. When the bony structures are being eroded, it will be a continuous boring pain. Often it is sharp, radiating or neuralgic, as it is reflected down the arm or along the intercostal nerves. This will vary in accordance with posture. The paroxysmal resemblance of anginal attacks is sometimes recognized. When the aneurysm develops suddenly as the result of severe strain, the patient is liable to complain of an excruciating pain with the sense of something suddenly "giving way." The extrinsic pain will vary as the pressure is relieved, and thus it will be observed that relief is experienced when the osseous erosion is complete and the softer tissues are not so resisting. Intrinsic pain is always lessened as the vascular tension is lowered.

Dyspnea is a prominent symptom and is more distressing than when it accompanies ordinary disturbances. It may become continuous and is subject to exacerbations. It is due to pressure upon the trachea, bronchi or a laryngeal nerve. The muscles of forced respiration are often excited and symptoms of spasmodic asthma may intervene. *Cough* is spasmodic and bothersome in its persistency. When the pressure affects the trachea, it is paroxysmal and dry in character. When the bronchi or a portion of the lung are impinged upon, causing a retention of secretions, it will simulate the bronchiectatic paroxysm and will give rise to a copious expectoration of thick and ropy muco-pus, which settles in layers. It has a toneless and muffled character when the vocal chords are affected. A hoarse cough or spasm of the vocal chords follow the slight compression or irritation of the recurrent laryngeal nerves, while a complete compression results in a paralysis which is recognized by aphonia. *Hemoptysis* is an occasional occurrence, secondary to granulations on the tracheal mucosa, to bronchial congestion, necrosis of lung tissue or a gradual leakage of the aneurysm. *Dysphagia* is a symptom which has already been mentioned in connection with esophageal pressure, and involvement of the *sympathetic nerves* displays signs of pupillary changes and other nerve disturbances of various types.

Physical Signs.—*Inspection.*—Pulsation is the significant sign; it is expansile in character and may be forcible and heaving; if it is associated with any swelling, the diagnosis is more positive. It is observed most frequently in the right second interspace, and is rarely found in the corresponding area to the left of the sternum. When the tumor is located along the transverse aorta, the pulsation may be noticed at the episternal notch. When the innominate is involved, the throbbing will be detected on the right side of the neck above the sternoclavicular junction or just above the sternum toward the right. Should the aneurysm be located near the origin of the aorta, the pulsation may be as low as the left third interspace near the sternal border. When the descending portion is the seat of the tumor, it may be detected also as low as the third left interspace. Inspection may discover a pulsating tumor in the left scapular zone and spinal curvature is sometimes apparent. The swelling in some cases may be so slight as to be imperceptible, while at other times it represents a tumor of considerable size. The overlaying skin may be thinner or show signs of ulceration. The appearance of the patient in advanced cases is suggestive of emphysema. The barrel-shaped chest, the elevation of the ribs and sternum at each inspiration, together with the prolonged and labored expiration, are quite distinctive.

Palpation helps to confirm the expansile character of the pulsation. The tumor may be compressible and fluctuating to the sense of touch, or, in case of any thickening from the deposit of fibrin, a firm and resisting feeling is experienced. Bimanual palpation, with one hand on the chest and the other on the back, is necessary to determine the character of the tumor. A diastolic shock succeeding the systolic impulse is a valuable sign, and tracheal tugging may often be revealed to the hand. *Percussion* does not always give conclusive evidence. As the aneurysm develops there may be sufficient retraction or collapse of the neighboring lung tissue to give localized flatness on deep percussion. A sense of increased resistance is also experienced. This flat sound will be detected to the right of the sternum when the ascending arch is involved, and in the left interscapular or scapular area when the descending portion of the arch is involved. When the aneurysm is located in the transverse aorta, dulness will not be so easily discovered. Pressure displacement of the heart will change the area of cardiac dulness. *Auscultation* gives positive or negative signs in proportion to the size, location and pathologic state of the tumor. Much depends upon the amount of coagulum in the sac. A systolic bruit is frequently heard over the seat of the tumor and is transmitted into the vessels of the neck. A diastolic murmur may exist alone when the lesion is near the aortic ring, or there may be a modification of both sounds. The bruit is generally defined as loud or booming. The heart sounds may be heard over the tumor. It is safe to say that in the majority of cases no distinctive sound will be recognized. The pulse beyond the aneurysm will be lessened in volume and retarded. If the innominate is involved, the right pulse will be weak and retarded, while the left will be normal. On the contrary, we shall find a weak and delayed pulse in the left wrist should the transverse arch beyond the innominate be involved. The sphygmographic tracing will show a slanting upstroke with a broad summit and obliterated waves in the downstroke.

Diagnosis.—To accurately diagnose aneurysm is not an easy matter; the small and deep-seated tumors give such slight symptoms that it is often an impossibility. The antecedent history as given under the etiology must be carefully studied. The predisposing causes already enumerated must be carefully regarded and the pressure signs will demand close study. The presence of a pulsating tumor in the vicinity of the blood vessel is the most suggestive sign. Several differentiations must be made. *Mediastinal tumors* are often confusing. These, however, do not manifest such decided and expansile pulsation, unless the growth is very large; the systolic and diastolic shock are not marked; tracheal tugging is absent and the retarded pulse is not found. Pain, enlargement of the cervical or axillary glands, together with the characteristic cachexia, are invariably present. *Pulsating empyema* will give a different history, the pressure symptoms are not the same and the area of dulness is not limited to the aortic region. The cardiac shock is not discovered and the expansile pulsation is not present. *Neurotic pulsations* are more general, and intimate association with neurasthenic conditions is usually found. Paroxysmal tachycardia is a prevailing symptom not common in aneurysm. *Aortic regurgitation,* with dilatation of the ascending aorta, strongly simulates aneurysm of the arch. It does not, however, exhibit the signs of a tumor with expansile pulsation and other pressure symptoms. The compression of a bronchus by a large aneurysmal tumor may give rise to bronchiectasis or possibly to an abscess with septic sequelae suggestive of pulmonary tuberculosis; in this case, however, the sputum examination and other pulmonary findings should differentiate. Displacement of the heart, lung retraction or even spinal curvature may cause abnormal pulsations, but these should not be confused with aneurysm.

Prognosis.—Sudden death is a not infrequent termination and is due to rupture of the sac and hemorrhage into the heart, pericardium, esophagus, a bronchus, etc. Gradual bleeding from the sac may be followed by chronic heart affections or lead to a slowly-developing asthenia. Spontaneous recovery has been reported and there are instances in which the sac has been protected by fibrinous infiltration. With the greatest care life may be prolonged in some cases, but the prognosis must be regarded as extremely unfavorable.

Treatment.—Treatment does not offer much encouragement. The possibility of a cure depends mostly upon the coagulation of the blood within the sac. Any measure which favors this will have some remedial effect. This theory is confirmed by the tendency toward a spontaneous cure which is sometimes observed in the saccular variety with a narrow opening.

Enforced rest will contribute to this end more than any other agent. Emotional excitement, nerve tension, alcoholic and other stimulation must be eliminated. The heart must be kept in perfect repose and any tendency to palpitation must be overcome by remedies, and, if necessary, by means of the icebag. Cardiac complications should receive careful treatment. A considerate solid diet will also favor the blood coagulation. Venesection has been claimed as a temporary relief, but in all probability it is at best only temporary and certainly will not do in anemic cases.

The use of remedies has not given much encouragement. It has been claimed that *potassium iodid*, by its internal action, has a tendency to thicken and contract the aneurysmal wall, and also to decrease the blood pressure. When syphilis is a factor, it is, no doubt, worthy of consideration. Maximum doses should be attained by degrees. Other preparations of iodin—particularly those which cause least gastric disturbance—may be used with some measure of success. If any results are to be obtained in this way, the treatment must be followed for many months. *Ergot,* or better, *ergotin,* has also been recommended to contract the sac and yet the unfavorable features of this remedy would naturally discourage its use, except in extreme cases. The aqueous solution has been injected into the sac with varying results. *Acupuncture,* by means of an iron wire or needle introduced into the sac, has been employed with the hope that coagulation of blood upon this foreign substance would to some extent favor contraction. *Galvano-puncture,* wiring and electrolysis have also been advocated and astringents, such as the solution of lead or persulphate of iron, have been injected for the same purpose. The unfavorable results and the incident dangers have condemned these efforts as too heroic and uncertain for safe practice. The subcutaneous injection of *gelatin* has received considerable attention of late. Two parts of gelatin are dissolved in one hundred parts of normal saline solution. Two hundred and fifty cubic centimeters of this are injected under the skin in the region of the thigh. About a dozen injections are necessary for the cure. We have no positive statistics confirming the value of this procedure. *Ligation* of the carotid and subclavian arteries has also been suggested.

Few physicians will resort to the above expedients unless some demand for heroic effort is made. Experienced practicians will always realize the almost inevitable failure of such measures. Peripheral aneurysm will doubtless yield somewhat to compression, but the control of violent heart action by the use of cardiac sedatives will accomplish equally as much. The nervous system must always be kept under proper subjection and remedies should be prescribed on the general principles of symptomatic suggestion. It may be best and necessary to relieve pain by the use of morphin, but the remedies mentioned under the general treatment of heart disease must always be taken into consideration. Homeopathically we may look for help from *aconite, digitalis, gelsemium, veratrum viride* and such constitutional remedies as *plumbum iodid, calcarea, ferrum phos., arsenicum iodid, spongia* and *kali iodid.*

ANEURYSM OF THE ABDOMINAL AORTA.

The neighborhood of the celiac axis may be the seat of aneurysm, a variety of forms being possible, though the most common are the fusiform and saccular. Occasionally there are multiple formations. If the tumor presses upon the vertebrae erosion may occur and the spinal cord may be compressed, causing paraplegia, with its peculiar train of symptoms. Development anteriorly is more common. Rupture into the lungs or pleurae is a possibility in case the diaphragm is perforated.

Pain is an early and persistent symptom when the nerve trunks are irritated by pressure. At first this may be lancinating in character and extend toward the umbilicus or downward into the extremities. As pressure and erosion increase the pain assumes a boring feature, and later is liable to become paroxysmal and colicky. Gastric disorders and hepatic hyperemia may give rise to vomiting and jaundice; other disturbing pressure symptoms may be present.

Physical Signs.—A *pulsating tumor,* expansile in character, is usually recognized by inspection or palpation. If the tumor is attached to the diaphragm the cardiac beating may give evidence of a double pulsation. The femoral pulse is usually diminished in volume and somewhat retarded. The presence of the tumor is observed most frequently just above the umbilicus and to the left of the median line. It may also project toward the left hypochondrium or the lateral and posterior lumbar areas. It is often not palpable and particularly when it is just beneath the diaphragm. A soft systolic bruit is generally heard over the tumor; a diastolic murmur is possible.

Diagnosis.—The presence of a swelling over the course of the aorta, the expansile pulsation and the systolic bruit are characteristic signs for careful and comparative study. Neurotic pulsations will often be confusing, but the general features of neurasthenia and the absence of a tumor will aid the decision. Solid tumors which press upon the aorta will be recognized when the patient takes a position which allows the tumor to fall forward away from the blood vessel. Anemia will be differentiated by the blood examination and other typical symptoms.

Prognosis and Treatment.—We should not offer much encouragement in the treatment. Spontaneous obliteration may be hoped for as in other forms of aneurysm, but is rare. Rupture is the constant danger. Cord compression, paraplegia and embolism of the superior mesenteric artery are also to be feared. Treatment gives little hope and in general it should be similar to that employed in thoracic aneurysm. The opportunities for employing the heroic measures suggested under that head do not, however, present themselves so favorably.

ANEURYSM OF THE PULMONARY ARTERY.

Aneurysm of the pulmonary artery is not common, though there are frequent dilatations of this vessel as a result of obstruction in the pulmonic circulation. Disease of the mitral valves, emphysema and tuberculosis contribute frequently to this condition.

Symptoms and Physical Signs.—Sufficient diagnostic signs to define the presence of aneurysm of the pulmonary artery are not always present. Disturbances of pulmonary circulation, dyspnea, cyanosis and cough may furnish the only suspicious features prior to the autopsy. There may be some pressure symptoms similar to those of aortic aneurysm. Pulsation may be discerned in the second left interspace and palpation may find an appreciable thrill and diastolic shock. A superficial systolic murmur is sometimes an attending physical sign in the same area. Some dulness is usually discovered at this point. The right ventricle suffers slight enlargement.

The prognosis is always unfavorable. The treatment is the same as that given in the preceding chapter.

Aneurysms of the celiac axis.—Aneurysm of this part of the aorta does not frequently occur, but it has been observed in connection with a similar condition in the upper part of the abdominal aorta.

Aneurysm of the splenic artery occasionally exists in the single or multiple form, but does not attain much size. Deep-seated and radiating abdominal pain, vomiting, the systolic bruit and the expansile pulsation are the diagnostic symptoms. A tumor may be at times detected in the left hypochondriac area. As hematemesis is sometimes present, gastric ulcer may be suspected. *Aneurysm of the hepatic artery* is unusually rare and is characterized by colicky pains below the epigastrium, vomiting, hematemesis and jaundice. Pulsation may be detected, but the presence of a tumor is not easily found. *Aneurysm of the superior mesenteric artery* is of exceptional occurrence. When it exists it will be demonstrated by epigastric and lumbar pain and a typical tumor near the abdominal median line. Mesenteric embolism and peritonitis following rupture, are the possible serious consequences. *Aneurysm of the inferior mesenteric artery* and *aneurysm of the renal arteries* are too rare to permit of any extended description. Congenital aneurysm is not a common discovery, though it may appear as the result of maldevelopment of the elastic coat of an artery during the ante natal period. The multiple form is most common, but the tumor is comparatively small in size. The aneurysmal wall consists of the adventitia and the intima alone. A form of congenital aneurysm has been described by Eppinger as *periarteritis nodosa,* in which nodules of inflammatory character have been found on the outer coat. This is, of course, found only among children.

DISEASES OF THE DIGESTIVE SYSTEM.

DISEASES OF THE MOUTH.

STOMATITIS.

Stomatitis is an inflammation of the mucous membrane of the mouth and appears in five forms: Catarrhal, aphthous, parasitic, ulcerative and gangrenous.

Catarrhal Stomatitis.—This is the mild form of stomatitis and is commonly found in children. It is a simple catarrhal inflammation which affects the mucous membrane of the mouth without any destruction of the deeper tissues. Chemical and mechanical irritants commonly create the inflammation; it may result from over-heated food, acids, alkalies, tobacco and stimulants. With children imperfect dentition, poor food and malnutrition are predisposing factors. Improper care of the mouth, decomposed food and infection should be considered in both children and adults. The eruptive fevers, gastro-intestinal disturbances and direct inflammatory extension from the tonsils and pharynx are exceptional causes.

The *symptoms* consist in *marked redness* of the buccal mucous membrane and the secretions of the mouth are generally decreased at first but later they are increased in quantity. The *swelling* may extend to the mucous membrane of the cheeks and lips while the furred tongue shows the indentation of the teeth. *Pain* is caused by the introduction of food, and the suckling child will not take the nipple. *Restlessness* and slight febrile movement will usually be noticed. The *dribbling saliva* is acid in reaction and the microscope shows the presence of desquamated pavement epithelium, leukocytes and micrococci. The inflammatory invasion is acute in character, the duration is short and the prognosis is usually favorable.

Aphthous Stomatitis.—This form is included under various designating terms. Vesicular, herpetic and follicular stomatitis are synonymous terms. The vesicles appear on the lips, cheeks or edges of the tongue, are small and round, rapidly degenerating into yellowish-white spots surrounded by red areolae.

Though adults may be afflicted, children are more frequently attacked by it. The spring and autumn furnish the greater number of cases, and there is no doubt that the acute exanthemata are most frequently the direct cause. Malnutrition, anemia and gastro-intestinal disturbances have something to do as predisposing factors. We may frequently observe it in association with the herpetic eruptions attending pneumonia. Forcheimer claims that the exciting cause may be found in "deleterious substances in the circulation which are bacterial or toxic."

Symptoms.—The aphthous ulcer follows the rupture of the vesicle; *pain* is, therefore, a common symptom. The *vesicles* may appear after the prodromal fever, though there may be no constitutional disturbance. In the beginning it resembles catarrhal stomatitis. The *saliva* is not greatly increased and the breath is rarely offensive. The aphthae, which follow specific infectious fevers, may form large irregular ulcers which coalesce and give a confluent form. *Riga's disease* is a form in which the frenum of the tongue is covered by a pearly indurated membrane. It is found in unhealthy and cachectic children and occurs often as an epidemic.

The *discrete form* is mild in character, runs a short course of not more than three or four days and responds quite readily to treatment. The *confluent form,* however, is very persistent and troublesome. Unfavorable relapses and recurrences are liable to take place.

Parasitic Stomatitis.—This form of the disease is described also under the terms "thrush" and "mycotic stomatitis." It is characterized by the rapid formation of grayish-white deposits on the buccal and pharyngeal mucous membranes, which have a tendency to coalesce and cover the entire mucous membrane. The exudate is a contagious fungus of the yeast variety and is known as the oidium albicans or saccharomyces albicans. The formation is not always confined to the buccal cavity; it may in severe cases extend even to the esophagus and larynx. The areas of the specific exudate are surrounded by an inflammatory areola giving rise, during the progress of the disease, to excoriation and bleeding from the mucous membrane.

Etiology.—"Thrush" is a disease found most frequently among children who are nursed from the bottle. The *lack* of *cleanliness* in the care of the bottle and nipple is the most common source of infection. *Unhygienic surroundings* and disorders of the gastro-intestinal tract always favor its development. The *exanthemata,* congenital syphilis and tuberculosis have been considered as predisposing causes. In fact any inherited disease, or the cachectic state following low grade fevers, may be looked upon as possible factors in its development. The *fermentation* of saccharine or impure food not only acidifies the alkaline secretions of the mouth but impairs nutrition of the mucosa. This condition favors the fungus growth which also adds to the unhealthy acid reaction. The disease may be observed in adults complicating diabetes mellitus, tuberculosis and other debilitating diseases. *Mechanical injury* of the oral mucous membrane and catarrhal stomatitis also favor this infection.

Symptoms.—The *early symptoms* are not always indicative of the disease. Aside from the local symptoms there are not generally many suggestive signs of physical disturbance unless we notice gastro-intestinal disorders and an inability to take food. *Pain* during the act of nursing is evident, and heat and dryness of the oral mucous membrane may be recognized. Upon examination of the mouth the *thrush spots* will be found. They are slightly raised above the surface and are found on the tongue, cheeks, lips and hard palate. In rare cases they may develop on the tonsils, pharynx and esophagus. We have every reason to believe that the stomach is also invaded by the fungus. While these spots are at first *isolated,* later they *coalesce* and may change from the pearly white color to a yellow or brownish *color* owing to the change in the secretions. In the early stages the exudate is *adherent* but later it is easily *detached,* leaving a smooth surface. There may be *successive crops* of the thrush spots and in some instances the deeper tissues are invaded. *Other fungi* may develop in conjunction with the typical exudate of thrush as the warmth and moisture of the mouth favor such processes. *Ulceration* of the oral mucous membrane is a possible sequence as the fungi are detached. *Diarrhea* may follow the introduction of this foreign matter into the intestinal tract.

Diagnosis.—Microscopic examination will reveal the cause of the disease, though it is possible to find the thrush organism in connection with other false membranes. *Milk curds* should cause no uncertainty in diagnosis as they are so easily removed. From *aphthous stomatitis* we shall find our greatest difficulty in differentiation. Yet in this disease salivation takes the place of the dryness of the mouth and a more pronounced ulcer of whitish-yellow color is surrounded by a red areola; when this is removed by the forceps bleeding is more liable to follow. Again these spots are not numerous and are generally discrete. They are very tender and appear at first as herpetic vesicles. The most distinctive difference is found in the absence of micro-organisms.

The *prognosis* is invariably favorable unless there are complicating diseases or a general cachectic condition which of course retards the recovery. The average case usually responds to treatment in a few days.

Ulcerative Stomatitis.—This form of stomatitis has other names such as stomacace, fetid stomatitis, and putrid sore mouth. It is a more serious disease as it is attended by mucous membrane necrosis and more pronounced ulceration.

Etiology.—Any of the other forms of stomatitis may predispose to this. It is essentially a disease of late childhood or early adult life and abrasion of the oral mucous membrane is invariably the starting point. Thus the use of the tooth brush, or a carious tooth may create the initial lesion. The lack of personal hygiene, impure food, contagious and debilitating diseases are all contributing factors. Impetigo contagiosa, scurvy, mercurialization, lead and phosphorus poisoning are also looked upon as possible causes. *The exciting cause* is no doubt an infectious organism not yet known though the streptococcus and staphylococcus are presumably present.

Symptoms.—The *ulcers* vary in size, are of an ashen gray color with red areola and show a tendency to bleed easily. They appear at first at the edges of the gums opposite the lower incisors and spread to the adjoining parts of the lips, cheeks and tongue. The *gums* become spongy and may bleed excessively. In extreme cases the *teeth* are liable to be loosened and the alveolar periosteum may undergo necrosis. *Profuse salivation,* pain and tenderness and the fetid odor of the breath are characteristic symptoms. The *tongue* is coated, swollen and shows the imprint of the teeth; the submaxillary glands are liable also to be swollen. As the *constitutional disturbances* become extreme, fever is a possibility—and a lowered state of vitality will give rise to cachexia and other symptoms of debility. Gastric complications will be recognized by the nausea and vomiting while an offensive diarrhea may follow as the result of swallowing the putrid discharges. The disease usually follows an acute course, though in rare exceptions it may assume a chronic character.

Mercurial Stomatitis, or mercurial ptyalism, is a form of the disease which is induced by the internal administration of mercury or when absorption follows its handling in the course of an occupation. The symptoms are strikingly similar, as ptyalism, fetor of the breath, swelling of the oral mucous membrane and ulceration of the gums are observed. Loss of the teeth and alveolar necrosis are natural results.

Syphilitic Stomatitis is likewise ulcerative in character but the ulcers are found also in the throat and at the angles of the mouth. They do not bleed as freely as the non-specific ulcers but the tissue degeneration is deeper. *Parrot's ulceration* is a form which occurs in the new-born and it is characterized by the symmetrical appearance of ulcers on the hard palate on both sides of the median line. *Bednar's aphthae* are two symmetrical ulcers on either side of the median line of the hard palate. They are of traumatic origin and usually may be traced to the pressure of an artificial nipple. Both of these latter diseases are innocent in character and generally heal in a few days unless the mouth is allowed to remain unduly filthy. *Neurotic ulceration* is a form of stomatitis described by Jacobi. It is an herpetic eruption which occurs invariably in neurotic subjects. If the neurasthenic condition is extreme the disease is liable to persist for a long time.

The *diagnosis* of ulcerative stomatitis should not be difficult when the characteristic symptoms just mentioned are clearly discerned. The *prognosis* is always favorable unless the ensuing debility is extreme.

Gangrenous Stomatitis.—Cancrum oris is the common term applied to this disease though the other synonyms, water cancer and noma, are equally recognized by the profession. The disease is rare and is characterized by an inflammatory infiltration of the cheek, near the angle of the mouth, which undergoes a rapid gangrenous degeneration. The cheek may be perforated and sometimes the process starts in the gums and ends in gangrenous necrosis. It is generally asymmetrical. The induration and sloughing develop rapidly and may extend in all directions from the original focus.

Etiology.—This is essentially a disease of the *poorly nourished,* occurs mostly among children of faulty heredity and is most conspicuous in hospitals and institutions where sanitary and hygienic conditions are not perfect. It rarely affects adults. The *infectious fevers* of childhood are the most common predisposing causes though it may be regarded as a primary disease. Climatic conditions are no doubt of some influence as noma is so often found in damp localities. *Ulcerative stomatitis* and other local irritations are regarded as factors favorable to its development. *No specific organism* has as yet been found, though the Klebs-Loeffler bacilli, staphylococci and streptococci have been discovered and therefore it is presumed to be due to a mixed infection.

Symptoms.—The *clinical course* of the disease is often insidious until the gangrenous process is once established and then it develops rapidly. The *gangrenous odor* of the breath and the saliva may be the first suggestive symptom. An early examination may reveal a blister or diffuse induration but usually the sloughing tissue first attracts attention. *Pain* is not a common complaint. The repulsive odor of the breath becomes rapidly worse and the dead tissue comes away in shreds. The *constitutional symptoms* are marked as the local destruction of tissue increases. High fever, a frequent pulse and rapid exhaustion are soon recognized. *Adjacent lymphatics* are soon implicated, and gastro-intestinal irritation may follow the swallowing of the poisonous discharges.

Diagnosis.—No other disease is liable to be confused with this unless it be *malignant pustule* and *ulcerative stomatitis.* The former is defined by its bacillus and its invasion is not so local. The latter does not show such rapid and extreme degeneration of tissue and rarely invades the external integument.

The *prognosis* is always unfavorable as the patient dies from exhaustion and sepsis in 80 to 90 per cent of the cases.

The General Treatment of Stomatitis.—There is so much in common pertaining to the treatment of all varieties of stomatitis that the subject may be condensed under a single head. In the first place the most important feature of the treatment must essentially relate to *cleanliness of the mouth.* This will necessitate clean and nourishing food, the proper care and cleansing of the mouth and the sterilizing of the nursing bottle and other eating utensils. At all times we must *guard against infection* and when it once exists antiseptics should be resorted to until the mouth is in a healthy condition. In children prophylaxis is most important for the oral mucous membrane is susceptible to irritants and infections. For this reason the mouth should be washed after every nursing.

The *prescriptions for local treatment* will vary in accordance with the character and extent of the stomatitis. Weak solutions of *boracic acid* or borate of sodium are sufficient for the milder cases. *Chlorate of potash, sulpho-carbolate of soda, listerine, borolyptol* and other bland washes answer for the average case. When stronger astringents are demanded solutions *of tannin, sulphate of zinc, silver nitrate,* etc., may be indicated. These, however, should be used with utmost caution as the reaction from too strong an astringent may retard the recovery. We may accept the rule that it is best to destroy any adventitious tissue by a powerful escharotic, rather than to allow an increase in the infection. *Carbolic acid* (2-5 percent) solution, the tincture of *myrrh* or *guaiac, peroxid of hydrogen,* and *permanganate of potash* are useful agents in many cases. The *glycerite of tannin, alum* and equal parts of *myrrh* and *rhatany* may be painted on the ulcerated spots. Hot milk with lime water, mucilaginous decoctions and soda solutions will often suffice as local palliatives in apparently severe cases. The *wine of opium* or *cocain* (4 per cent) may be necessary when pain is a prominent factor.

In the *aphthous ulcers* it may be necessary to touch the parts with ninety-five per cent *carbolic acid* or the solid *nitrate* of *silver,* and then resort to the milder washes in subsequent treatment. *Gangrenous tissue* will demand a more energetic escharotic hence it may be best to use the actual cautery or such strong caustics as *nitric acid,* the *acid nitrate of mercury, solid zinc chlorid* or *Vienna paste.* Other astringents and disinfectants will be indicated in accordance with the symptoms and the gravity of the disease.

The *general treatment* must consider the constitutional symptoms, the diet and the care of the patient in accordance with the best principles of practice. Anemia, malnutrition and general debility will necessitate the closest care. Remedies which correct these perversions will give the best results. In the majority of cases it may be well to consider the use of the iodids.

Borax is one of our best internal remedies. It refers in general to the aphthous oral ulcerations and the gastro-intestinal irritation. *Mercury* in potency covers the same symptoms and also refers to the salivation, the indented tongue, the swollen condition of the mucous membrane and the glandular perversions. In a measure *hepar sulphur* and the *iodid* of *arsenic* have a similar usefulness. *Sulphur* points to a more pronounced systemic disturbance and the same may be said of *phosphorus* and *pulsatilla.* *Nitric acid* and *argentum nit.* cover the symptoms pertaining to well developed ulceration and the invasion of deeper tissues. *Arsenicum alb., muriatic acid* and *calcarea carb.* will be indicated when this perversion is not so severe. *Phytolacca, thuja* and *hydrastis* will correct the inflammatory condition when it tends to be chronic. For the more pronounced inflammation we may consider such remedies as *baptisia, ailanthus, arum triphyllum, echinacea* and *belladonna.* Other remedies will be suggested in accordance with their indications.

DISEASES OF THE TONGUE.

GLOSSITIS.

Acute Glossitis.—This is an acute parenchymatous inflammation of the tongue. It is not a common disease and occurs as the result of traumatism, burns, scald, poisonous stings and corrosive irritants. Latent causes either of microbic or systemic origin are often fundamental.

Symptoms.—These develop rapidly. The *tongue* is swollen abnormally and may protrude beyond the lips. *Speech,* mastication and deglutition are performed with difficulty. *Respiration* is also greatly impaired and dyspnea is sometimes a mechanical result. When the tongue is exposed it becomes dry and fissured. *Suffocation* is not an uncommon termination. In association with acute glossitis we may frequently observe a catarrhal stomatitis and in such cases salivation is usually profuse. *Moderate fever* may be observed and glandular inflammation is a common sequence. The inflammatory invasion attains the maximum in three or four days and the subsidence occurs with gradual relief of local symptoms.

The *prognosis* in the majority of cases is favorable and the *treatment* is usually of the simplest character. *Locally* the use of ice and mucilaginous mouth washes are usually sufficient. Alkaline solutions and mild antiseptics are about all that are necessary in the majority of cases. Abscesses will of course demand incision. Such remedies as *aconite, belladonna, rhus tox* and *bryonia* will generally answer for the febrile condition. *Apis, arsenicum, lachesis, hepar sulphur, mercury* and *iodid of potassium* will be suggested in accordance with their indications.

Chronic Superficial Glossitis.—This is a chronic inflammation of the mucosa of the tongue. It follows acute attacks or it may be incident to the extreme use of tobacco or stimulants. Irritating food may also be the inciting cause. The surface of the tongue is red and sensitive and furrows or patches which signify inflammatory invasion are often seen. The general health is invariably impaired and symptoms of other disorders may be present. The *prognosis* is favorable so far as alleviation is concerned but the chronic state may persist for a long time. The *restriction of irritating foods* and drinks and the use of mild demulcents will generally suffice in the treatment. The indicated remedies and tonics which relieve the systemic depletion are about all that is necessary for the internal treatment.

Glossitis desiccans is the term applied to a rare disease of the tongue in which fissures and indentations are characteristic. The ulceration and excoriation may be so pronounced that extreme pain is a constant symptom. The disease will respond to those remedies and local washes which reduce the persistent irritation and inflammation. *Lingual psoriasis* is a disease of the tongue which is characterized by areas of hyperplasia of the epithelium known as "lingua geographica." The etiology is rather obscure and the disease may run a chronic course for years. *Buccal psoriasis* or leukoplakia oris is an affection of the mucous membrane developing in the form of white or bluish-white patches on the cheeks and lateral borders of the tongue. These often present a notched or scar-like appearance which resembles syphilitic excoriations. While the cause is unknown the specific taint is often predisposing. Outside of occasional pain the symptoms are not pronounced and it yields to remedies for local and specific indications. *Angina Ludovici,* or Ludwig's angina, is a disease of the mouth in which acute phlegmonous inflammation is the typical feature. Infectious diseases like diphtheria and scarlet fever are thought to be the primary causes. Traumatism and the extension of inflammation from the adjacent glands are possible causes. The symptoms are pronounced; swelling of the submaxillary glands and the cellular tissue is noticed at first. Pain is excessive and articulation, mastication and deglutition are decidedly impaired. Constitutional disturbances are recognized in the low-grade fever while extensive sloughing is not an uncommon experience. The outcome is uncertain and the treatment will be symptomatic in accordance with the degree of infection.

DISEASES OF THE SALIVARY GLANDS.

PTYALISM.

Hypersecretion and salivation are the other terms used referring to an abnormal increase in the flow of saliva. There are various causes which produce this condition. As an idiopathic affection it is rare, and nervous excitement may be mentioned as the inciting cause in such instances. The prolonged and indiscriminate use of mercury in massive doses is no doubt the most frequent cause, though we do not see so many cases from this nowadays, as the profession has greatly reduced the dosage of this drug. It may also be a symptom of disease of the mouth—for instance, accompanying noma or ulcerative stomatitis. Gastro-intestinal and pancreatic disease, or diseases of the genital tract may incite it, and other toxemias may be reckoned as possible causes. Tumors of the medulla which involve the nerve supply of the mouth are not uncommon causes of this condition. Parotitis and diseases which affect the gums, the irritation of carious teeth, and infections which pervert the oral secretions may be looked upon as possible sources of the disease.

Symptoms.—In the *early stages* the patient complains of tenderness of the teeth and a peculiar metallic taste in the mouth. The gums are red and swollen and recede from the teeth. The *flow of saliva* gradually increases and dribbles from the corners of the mouth. *The inflammation* may invade the whole oral mucous membrane and frequently terminates in an ulcerative process. *Gangrenous degeneration* of tissue may follow a protracted or severe case. Unless a drug excess is the underlying cause, the *prognosis* may be regarded usually favorable as to cure and the *diagnosis* is not difficult to settle.

Treatment.—The removal of the irritating cause and the correction of physical conditions which predispose to salivation are the features of treatment to be considered. Mild mouth washes such as *permanganate of potash, listerine, hydrogen, peroxid* and *soda solutions* are generally sufficient for local treatment. The internal remedies for obstinate cases will generally be such as *mercurius,* the *iodids, nitric acid* and *sulphur. Helonias, jaborandi, euphorbium, hydrastis* and *borax* may often be indicated.

XEROSTOMA.

As a contrast to hypersecretion we find that this disease is a condition of aptyalism or "dry mouth," as it is often defined. As a distinct disease it is rarely found and the causes are not well understood. Perversions of the nerve supply of the salivary glands are supposed to be the primary factors, and it may arise from the occurrence of mental shocks. Mouth breathing and febrile diseases are sometimes causative factors and diabetes may predispose.

The *symptoms* are a deficiency of saliva and persistent dryness of the mouth. Deglutition and articulation are performed with difficulty. *Locally* the mucous membrane presents a shiny, red or glazed appearance; at times the tongue may be cracked and the teeth may crumble. *Inspection* easily settles the diagnosis and the *prognosis* depends entirely upon the cause and the ability of the patient to respond to treatment.

Treatment is not very successful unless the primary cause is discovered and corrected in time. *Mild astringent* solutions will help in some cases while others may respond to the careful use of well selected remedies. *Iodid* of *potash* and *pilocarpin* have been recommended as standard remedies. Galvanism has been advised as an agent for stimulating the nerve impairment.

Glass-blower's mouth is another term applied to a similar condition. In this case, however, the cause is always known. The cheek muscles present a hernial bulging and the epithelium of the mucous membrane shows opaline plaques. The buccal mucosa is distended and the parotids are often emphysematous. Steno's duct may also be distended.

METASTATIC PAROTITIS.

The other terms for this disease are "symptomatic" parotitis or "parotid buboes." It is a septic inflammation of the parotid gland and is generally attended by suppuration. A *milder form* may arise from local traumatism, occlusion of the salivary ducts, and the extension of inflammation from neighboring parts. Acute infectious fevers are the more common primary causes. Genito-urinary diseases and injuries of the abdominal or pelvic structures may be the source of the infection. Peripheral neuritis with a facial paralysis is recognized as the origin in some instances. A *chronic form* of the disease is a possibility. In such cases, however, the source is more liable to be traced to lead or mercurial poisoning. It may also be secondary to mumps, inflammation of the throat or to chronic diseases such as nephritis.

Symptoms.—The symptomatology will simulate that of idiopathic parotitis though the tendency is more toward the suppurative character. The inflamed gland is harder and there is always a possibility of gangrene. There should be no confusion of this disease with the inflammatory involvement of the cervical lymphatics, which is generally bilateral. The *prognosis* should be favorable though the disease is a serious one; much depends upon the degree of suppuration, which may extend to the ear.

Treatment.—As this usually terminates in suppuration its treatment, therefore, comes within the domain of surgery. Cleanliness of the mouth and antiseptic measures are always to be employed. Locally mouth washes containing *calendula, boric acid, permanganate of potash, pyoktanin*, or a 50 per cent solution of *peroxid of hydrogen* are serviceable in mild cases. The application of ice bags to the swollen surface may reduce the pain. The internal remedies which pertain to conditions of inflammation and suppuration should always be administered. *Belladonna, rhus tox, baptisia, pulsatilla* and *echinacea* are often indicated. When the suppuration appears we have no better remedies than *mercurius, hepar sulphur, silicea, arsenic, iodin* and *iodoform. Calcarea fluorica* should never be lost sight of in this respect.

DISEASES OF THE TONSILS.

ACUTE TONSILLITIS.

Inflammation which affects the tonsils gives rise to various forms of this disease; they are defined in accordance with the degree of local involvement and the symptoms. The following forms are recognized: (1) Acute catarrhal tonsillitis. (2) Acute follicular tonsillitis. (3) Acute parenchymatous tonsillitis. (4) Necrotic tonsillitis.

Acute Catarrhal Tonsillitis.—This is an acute inflammation of the mucous membrane which covers the tonsil. It is a result of exposure and is usually described as a catarrhal affection. It is of common occurrence in children, especially those of strumous diathesis. Retained concretions may be predisposing causes. It often exists where there is a chronic tonsillar hypertrophy, the acute feature coming as the result of a cold, especially during the spring season.

Symptoms.—The *local symptoms* of painful and difficult deglutition are early manifestations. Mild *constitutional disturbances* may be in evidence though temperature is not always observed. Local examination reveals marked redness and slight swelling of the mucous membrane covering the tonsil, perhaps associated with an acute pharyngitis. The dry and glazed membrane is later covered with a thin exudate which is easily detached. Uvular and faucial tumefaction are secondary conditions, and swelling of the cervical lymphatics may follow. The attacks come suddenly and ordinarily disappear in a day or two. Otitis media and acute pharyngitis may complicate. The *prognosis* is always favorable.

Acute Follicular Tonsillitis.—This is an acute invasion of the mucous membrane lining the tonsillar crypts. The surface of the tonsils is invariably affected by a coexisting catarrhal tonsillitis, and the tonsil as a whole is greatly swollen. While this affection may involve but one tonsil it is generally bilateral. The lacunae become filled with a cheesy exudate which protrudes upon the surface of the tonsil and is composed of epithelial and pus cells, cellular debris, and numerous bacteria. These masses may be pressed out of the follicles. Often their retention may lead to follicular abscesses. The lymphatics are generally involved.

Symptoms.—The *constitutional symptoms* are quite sudden and severe. A pronounced chill, headache, backache, anorexia and a temperature (102-105° F.) are the early signs. The depression is often so profound that it suggests a more serious infection but in the ordinary cases this passes away in a few days. The rheumatic diathesis may complicate the disease and in such cases the tonsillitis is liable to assume a chronic form. Calcification of the exudate is not an uncommon result in patients with depraved nutrition.

The *diagnosis* is not difficult on account of the location and appearance of the exudate. The severe cases may give the appearance of a diphtheritic involvement but this disease usually shows a tendency to spread to the palatine arches, velum or pharynx. The bacteriological examination will always settle this doubt. The *prognosis* is invariably favorable though recurrence is a possibility and subacute tonsillitis may continue for some time.

Acute Parenchymatous Tonsillitis.—This is otherwise known as tonsillar abscess. It occurs during the later periods of life and it is inclined to be of severe type. The *dryness of the throat,* the pain and the difficult deglutition of the other forms are more pronounced. A *viscid mucus* covers parts of the affected tonsils and the swelling may be aggravated to the point that dyspnea is a symptom. Both tonsils are enlarged and edema, induration and a bluish-red color are characteristic symptoms. The *surrounding tissues* may partake of this pathology and the submaxillary and cervical glands are generally implicated. As the *exudate softens* abscess formation follows. This condition is known as peritonsillar abscess or "quinsy." Rupture and resolution are then the natural result.

The *constitutional symptoms* are pronounced. The temperature reaches 104°-105° F. and the pulse increases to 120°-130 per minute. Chills may precede and persist and delirium may attend the constitutional excitement. The *duration* depends upon the speedy evacuation of the pus and the involvement of the surrounding cellular tissue. *Edema of the larynx,* paralysis of the soft palate and pharynx and a chronic enlargement of the tonsils are frequent sequelae.

Necrotic Tonsillitis.—This has been defined by Strümpell as a severe form which simulates diphtheria and can be differentiated only by microscopic examination. It is characterized by a grayish-white adherent membrane and necrosis which is limited only by the exudate. The inflammation and induration are not extensive, but the ulcer is liable to remain even after the slough has been removed. The *diagnosis* and *prognosis* will be settled only by a painstaking study of the case. *Diphtheria* and *scarlet fever* must always be kept in mind.

General Treatment.—In the *acute catarrhal form* we shall obtain prompt relief by means of the indicated remedies. *Aconite, arnica, belladonna, pulsatilla and merc. sol.* are our best remedies. In the *follicular* and *parenchymatous* varieties we should consider such additional remedies as *rhus tox, baptisia, ailanthus, ranunculus, guaiac* and *phytolacca.* Remedies which pertain to the deeper-seated inflammation will be called for in accordance with their indications. Among these we may favorably mention *mercurius iodid, kali bichromicum, hepar sulphur, apis, lachesis, ledum, calcarea* or *baryta carb.* and *kali mur.* Many prefer the internal administration of sodium salicylate, salol and the ammoniated tincture of guaiac. The function of all the eliminative organs should be increased. Iron tonics are also suggested for the anemia; opiates are advised for pain, while mercury and the iodids are commonly employed.

Local Treatment.—In the acute catarrhal variety a simple *soda* or *guaiac gargle* may be sufficient. *Hot milk* with a few drops of the *ammoniated tincture of guaiac* is very efficacious. *Mild antiseptic solutions* such as Dobell's solution, Seiler's tablets, borax, thymol, carbolic acid or potassium permanganate used in an atomizer are popular agents. The strength of these should be measured in accordance with the gravity of the case. For the relief of pain many favor the application of an ice bag to the throat. One of the best applications I have ever used for quinsy is guaiacol-vasogen. Hot poultices, such as flaxseed, mustard or cornmeal, sprinkled with spirits of camphor are now quite generally discarded. Equal parts of the tincture of the *chlorid of iron, glycerin* and *water* applied to the tonsil with a camel hair brush is a popular preparation. A 10 per cent solution of *cocain* may relieve the pain; *silver nitrate* (40 grains to the ounce) is advocated by many. The recent use of *adrenalin* has given favorable results in the treatment of the local inflammation. *Free scarification* is not lauded so much of late though a liberal incision is necessary when pus can be localized. The disease may become so serious that excision of the tonsils will be demanded in certain cases after the acute attack has passed in order to lessen the liability to future attacks.

CHRONIC TONSILLITIS.

This term refers to a chronic inflammation of the tonsillar tissue which results in hypertrophy with a fibroid or calcareous degeneration. The faucial tonsils and the nasopharyngeal tonsil (Luschka's) are the ones most frequently involved, though the lingual tonsils often undergo similar change.

Chronic inflammation of the tonsils may be due to repeated acute attacks, and is usually observed in those of the lymphatic habit and strumous diathesis. Heredity may be a factor, while the infectious diseases of childhood favor its development.

Pathology and Symptoms.—The enlargement is usually of gradual occurrence and takes place before puberty, rarely beginning after that time. The enlargement may be simply due to hyperplasia, but more often is a true hypertrophy. Later the structure may undergo fibroid or calcareous degeneration.

Symptoms.—Very often the faucial tonsils are involved secondarily to the nasopharyngeal (adenoids). When the former alone are hypertrophied the symptoms will be those of frequent attacks of acute tonsillitis, difficulty in swallowing, gastric derangements due to diverted salivary secretion, and the "wooden" throaty voice. The cervical lymphatics will usually be found enlarged.

When adenoids are present, mouth breathing results. Because the post-nasal obstruction prevents normal breathing through the nose, the difference in air-pressure on the hard palate and on the nasal floor results in the former being elevated, thus narrowing its arch as well as that of the alveolar process. Likewise are the accessory sinuses affected, remaining more or less undeveloped.

The *facial expression* of the adenoid patient is characteristic. Apathetic, vacant, staring, with short upper lip and mouth constantly open, these little folks can be recognized at a distance as "mouth-breathers."

Because of the mouthbreathing the air is not properly warmed, moistened and filtered, consequently the blood being improperly aerated can not properly nourish the brain and body. These patients are nearly always below par in school work, physical development and endurance.

Usually these patients have more or less *acute ear involvement,* with defective hearing. The sense of taste and smell are almost invariably impaired and in those with tonsillar involvement, a fetid breath is present, due to the cheesy deposit in the crypts. The constantly open mouth in these cases tends to retard the secretion of saliva, consequently the digestion is impaired and nutrition is not up to the demands of the growing body.

Diagnosis.—The physical examination will settle the diagnosis though the above mentioned symptoms are diagnostic. In the normal condition the tonsils do not project beyond the pillars of the fauces. Palpation externally and internally will give a proper estimate of the tonsillar increase. Hypertrophy of the pharyngeal tissue will be determined by the finger and by rhinoscopic examination.

Prognosis.—Ordinarily the disease is not a dangerous one. Its impairment of the general health, however, can not be decried. It is a constant irritant which encourages throat troubles and it may be of serious consequence in the case of diphtheria or scarlet fever. It certainly encourages acute infections in these structures.

Treatment.—Some cases of hypertrophic tonsillitis may correct themselves by a natural atrophy as the patient grows older. It is safe to say that the majority need excision as the only means of cure. Adenoids are rarely relieved in any other way. Attention to nutrition and aseptic conditions of the throat and mouth are necessary. The predisposing factors must be treated from a hygienic and medicinal standpoint. *Local* *treatment* for catarrhal conditions must be instituted and astringents applied to the tonsils may help somewhat. Such internal medication as *mercury, hepar sulphur,* the *iodids,* the *calcareas* and other indicated remedies are useful to a degree.

These patients after removal of the enlarged tonsils are greatly benefited by an out-door life and nourishing food. It is these patients who are especially helped by cod liver oil and the vegetable and animal fats.

DISEASES OF THE PHARYNX.

PHARYNGITIS.

Acute Catarrhal Pharyngitis.—This is an acute catarrhal inflammation of the pharyngeal mucous membrane and is commonly called acute sore throat or simple angina. It is a frequent disease in children and it is not rare in adults. Exposure to cold and sudden changes of temperature are exciting causes though constitutional defects invariably predispose. It is associated with the scrofulous, rheumatic and gouty diathesis. With some it is an idiosyncrasy. Chronic tonsillitis and acute naso-pharyngeal catarrh are many times predisposing factors. Epidemic pharyngitis is not an impossibility. A form of exanthematous pharyngitis occurs in conjunction with the infectious diseases, particularly scarlet fever. Streptococci are in evidence in the exudate.

Symptoms.—A sense of *dryness* and *soreness* of the throat is experienced on deglutition. The patient is inclined to constantly clear the throat on account of the irritation produced by the muco-purulent secretion. The throat feels full and stiff and when the inflammation invades the larynx hoarseness is noticed. Often deafness is a symptom when the Eustachian tube is affected by the inflammatory process.

Locally we shall observe a reddened and tumefied appearance of the pharynx, the anterior and posterior fauces, the soft palate and the tonsillar surfaces. The *mucous secretion* may be abundant and *pharyngeal ulceration* may be excessive in some cases thus increasing the local soreness and the constitutional symptoms. In the beginning *chills and fever* are present in proportion to the extent of the pathological conditions.

Diagnosis.—The local examination will explain the symptoms and decide the diagnosis. Inasmuch as the disease runs a short course, of three to five days, the *prognosis* is favorable.

Chronic Pharyngitis.—Following the acute form or as an independent affection we frequently find a condition of chronic inflammation affecting the pharyngeal and adjacent mucous membrane. It may be diffuse or circumscribed in character. When the mucous membrane alone is involved we speak of it as *catarrhal pharyngitis* and when it is confined to the follicles of the pharynx it is termed *follicular pharyngitis.* The pathologic change will be recognized as hypertrophic or atrophic in character according to the structural perversion.

Etiology.—It is a disease which is found most frequently during adult life though a persistent recurrence of the acute form may cause its appearance at an earlier age. The exciting causes are the overuse and strain of the voice as observed in public speakers. Irritations from the use of tobacco, the presence of adenoids and of nasal imperfections are possible causes, and gastro-intestinal diseases are alike predisposing. Constitutional disturbances arising from the gouty, rheumatic, syphilitic and tuberculous diathesis are frequently the agents which develop it.

Pathology.—The *mucous membrane* in the simple chronic form is reddened, thickened and overactive in its mucous discharge, marking the *hypertrophic* form. In the *atrophic* form the membrane is pale, thin and dry. The veins are dilated and tortuous. In the *follicular variety* the mucous glands are swollen and red, forming a "split pea" elevation on the posterior pharyngeal wall. This is due to the lymphoid hyperplasia and the retained secretions.

Symptoms.—Few cases of the chronic variety give rise to marked symptoms. The patient may complain simply of a slight dryness or tickling in the throat. A *short dry cough,* with a tendency to clear the throat, will be noticed, particularly in the morning. When the larynx is involved *hoarseness* will be noticed. In the *hypertrophic form* a thick catarrhal discharge will be found in the posterior nares and the sense of taste may be impaired. When the Eustachian tubes are affected hearing will likewise be perverted. The *atrophic form,* with its pale and shining membrane, will present fewer of these symptoms but the pain may be more pronounced.

Prognosis.—In rendering an opinion regarding the outcome of this disease much will depend upon the constitutional predispositions and the pathologic character. *Atrophic pharyngitis* is not easily cured and the *follicular form* does not readily yield to remedies unless the general health is brought to par. Much also depends upon the extent and duration of the hypertrophy. At best it will yield only to persistent and painstaking treatment.

Ulceration of the Pharynx.—The ordinary form of chronic pharyngitis does not tend toward ulceration. When syphilis, tuberculosis, diphtheria and infective inflammations are associated with the debilitated state, obstinate and serious ulcerations may exist. The ulcers vary in size and are of a yellowish white color. The tonsils are often involved in ulcerative extension. Pain during deglutition is the most characteristic symptom. Prostration, a weak pulse and slightly elevated temperature mark the constitutional disturbance.

Diagnosis.—It is necessary to distinguish the various forms of ulceration. In the follicular form the ulcers are small, superficial and are usually associated with chronic pharyngeal catarrh. *Syphilitic ulcers* are but slightly painful and are found in the posterior wall of the pharynx; when secondary they are superficial and often associated with mucous patches; when they are tertiary they are deep and punched out in appearance. The history of syphilis is usually discovered. The *tuberculous ulcer* is very painful, is usually located on the posterior wall and generally spreads over a considerable area. In appearance it is irregular, superficial and has a grayish base. Pulmonary tuberculosis may also be present. *Indolent ulcers* may occur as the result of lowered nutrition; they are insidious in their development and present few symptoms.

Prognosis.—The follicular ulcerations yield to careful treatment. *Tuberculous ulcerations* depend greatly upon the general condition; when this improves they respond to treatment. *Syphilitic ulcerations* will be affected by a careful course of the iodids and the ordinary indolent ulcers will improve as the general condition is improved.

Herpetic Pharyngitis.—This is an inflammation of the pharyngeal mucous membrane characterized by the presence of peculiar herpetic vesicles. It is otherwise termed membranous or aphthous pharyngitis. It is a sequence of exposure to cold and occurs most frequently during the damp and changeable weather of spring. It is prone to affect women and children and has a decided relation to diphtheria as it occurs commonly during epidemics of that disease.

Symptoms. — *Fever,* general malaise and soreness of the throat are the early signs. The pharynx displays *successive crops of vesicles* which appear on the mucous membrane. The tongue, soft palate and mouth are also involved. It simulates the aphthous form of stomatitis. These vesicles rupture in about twenty-four hours and leave a group of superficial circular ulcers. These may coalesce and as they are often covered by a false membrane diphtheria may be suggested. This form of pharyngitis may be associated with herpetic eruptions on other parts of the body.

Diagnosis and Prognosis.—The *diagnosis* is easily settled when the disease is observed during the herpetic stage. When the ulcers and the false membrane are first seen it may be so suggestive of diphtheria that a bacteriologic examination will be necessary. The *prognosis* is invariably favorable unless there are severe complications.

Phlegmonous Pharyngitis.—This term refers to a *suppurating inflammation* which affects the pharynx. It may come from a suppurating tonsillitis or quinsy or it may be due to an infectious phlegmon of the pharynx. In this case there will be signs of external swelling and the pharyngeal mucous membrane will tend toward rapid suppuration. Injury, the inhaling of scalding fluids, or the swallowing of corrosive poisons are put down as possible causes. The lodgment of foreign bodies is often sufficient to produce this form of inflammation.

Erysipelas may invade the pharynx and give rise to the usual symptoms of that disease. In such cases the infection from the streptococcus of Fehleisen will be determined and the erysipelatous involvement of neighboring tissues will also be discovered. The symptoms, in addition to those characteristic of pharyngeal inflammation, consist in the redness, swelling, edema, blisters and glandular involvement which accompany any erysipelatous involvement. *Gangrenous pharyngitis* may follow this or any of the pharyngeal affections. The black or greenish spots, which increase in size, are easily detected. The fetid breath, the great depression following the high fever, the cold extremities and feeble pulse will aid in making the diagnosis.

Symptoms.—The symptoms of peritonsillar abscess are ushered in by *constitutional symptoms* of violent character. A decided chill is followed by high fever. The pain in deglutition is marked and the affected parts swell rapidly and the tonsils are invaded. The mucous membrane is edematous and purple in color. A *mucopurulent secretion* is profuse when the abscess ruptures. Headache is intense and delirium may ensue. *Respiration* is embarrassed and the *prostration* is profound. The typhoid state is liable to develop rapidly.

Prognosis.—Much depends upon the course of the inflammation. If suppuration is rapid and localized, relief may come from its evacuation. In case there is infiltration of surrounding parts the result will be unfavorable. Acute septic phlegmon is nearly always fatal.

Retropharyngeal Abscess.—This abscess formation is due to a rare suppurative inflammation in the connective-tissue between the pharynx and the vertebral column.

Etiology.—It is most commonly found in children though it may occur in adults. Antecedent dyscrasiae, hereditary or acquired, together with bad hygiene and persistent chronic pharyngitis, are predisposing causes. Tuberculosis and syphilis are considered as factors. Traumatism, caries of the cervical vertebrae and acute infections are frequent causes, though it may be a primary disease.

Pathology.—The suppurative process is observed in the lymphatic glands posterior to the pharynx, which are numerous and easily disposed to inflammation. The structure of the pharynx favors the burrowing of pus and cases are recorded in which it has extended as far as the mediastinum. When spinal caries is the cause the infection is usually tuberculous in character.

Symptoms.—The onset is insidious inasmuch as symptoms are not generally observed until the abscess develops. Pain in swallowing and deep-seated pharyngeal soreness may be felt at first. Later the throat feels stiff during deglutition. Respiratory embarrassment to the extent of stertorous breathing, and dyspnea are occasional symptoms. Cyanosis and convulsions have followed such conditions. A distressing cough is often present and aphasia is sometimes observed. The constitutional symptoms are characterized by chilliness, slight fever and languor. In adults rigors are common and the patient invariably complains of headache and digestive disturbances. Local examination reveals the presence of a tumor and palpation detects the fluctuation.

Prognosis.—If the diagnosis is made early immediate relief will follow free evacuation of the pus. Suffocation may take place should there be a rupture into the larynx. Unfortunate results always follow the burrowing into the dense pharyngeal tissue as the abscess may open into the esophagus or mediastinum. The disease is often mistaken for edema of the larynx and croup.

The General Treatment of Pharyngitis.—The *early symptoms* which point to sore throat or the inflammation of the pharynx should be vigorously treated. In this way we may avoid the sequelae which lead to abscess formation. Proper attention directed to hygiene, diet and the physical resistance will overcome the tendency to take cold which always favors pharyngitis. The *diathetic state* which may refer to tuberculosis, rheumatism or lithemia should be treated in accordance with the conditions which relate to those diseases.

The *local treatment* may be varied according to the degree of inflammation. In the acute catarrhal form much relief may be obtained by a cold compress or a small ice bag over the throat. The counter irritation of poultices is not so commonly used as in the past. Steam inhalations and gargles of hot soda water and other mild astringents are agreeable sedatives. Alum, or chlorid of iron—a teaspoonful to a glass of hot water—is very serviceable. Painting the pharynx with equal parts of glycerin and chlorid of iron will suffice in mild cases. Sprays of albolene or oils medicated with menthol and other drugs are agreeable applications and some advocate the use of a 2 per cent solution of cocain in menthol and albolene. Eucain may take the place of cocain, while adrenalin chlorid is probably better than any of the above in highly congested states. Dobell's solution and silver nitrate (10 grains to the ounce) are still employed with success. Insufflation with equal parts of powdered tannin and alum is an old expedient. The various sprays, douches and local treatments for nasal catarrh are valuable adjuvants. Enlarged follicles will require the use of the galvanic or actual cautery or chronic or trichloracetic acid. Adenoids should be removed surgically.

When *pus is present* the only relief will come from a free incision. In retropharyngeal abscess this must be performed as soon as fluctuation appears. The incision should be made in the median line and to prevent the pus from entering the larynx, the head should be inclined forward.

The use of *internal medicine* is·a matter of great importance. In this respect we have many homeopathic remedies which have served us well. *Belladonna* is probably the most useful in the early stages as it corresponds to many of the local and constitutional febrile manifestations. The dryness and redness of the throat, the constrictive sensation and pain during deglutition, the peculiar facial flush and the throbbing headache with the fever will readily lead to its selection. *Aconite* may be demanded in the incipient stages when the local and general symptoms are more intense. The former remedy applies also when the tendency is toward the phlegmonous form of pharyngitis. *Apis* is indicated when edema is pronounced and the throat is hot and dry but with little or no thirst. *Pulsatilla* has some of these symptoms but the affected area is usually covered with mucus. When the throat is dry and cracked and shows an inclination toward ulceration *arsenicum, baptisia* or *rhus tox* may be suggested. The peculiar burning and stinging, observed under apis, may also point to *cantharis* or *capsicum.* Pain, rawness and the swollen condition of the throat which makes swallowing difficult will be helped by *lachesis.* The left side is chiefly involved. *Kali bichromicum* relates to a deeper seated inflammation and the more tenacious mucus. *Causticum, nitric acid, naja, lycopodium* and *sulphur* may be compared in this connection. *Phytolacca* with its persistent soreness and deep faucial congestion, *sanguinaria* in the indurated swelling and *petroleum* in irritating inflammation are worthy of consideration.

In the chronic forms of pharyngitis we shall find our best help in *mercury biniodid* when the inflammation is associated with degeneration of tissue or organized exudation. *Argentum nitricum* is indicated by similar symptoms and the dryness of the throat gives the feeling of a splinter or some irritating foreign substance. When hoarseness is persistent *argentum metallicum* will be thought of. *Kali chlor.* and *kali iodid* and *hydrastis* are indicated by the presence of a tenacious mucus which characterizes chronic or indolent pharyngeal catarrh. *Arum triphyllum* is of value in the follicular inflammation. *Ailan-*

thus may be considered in this same sense. Other useful remedies are *arsenicum iodid, hepar sulphur, iodoform, selenium, stannum iodid* and *silicea.*

DISEASES OF THE ESOPHAGUS.

Methods of Examination.—Examinations of the esophagus are so important in the making of a diagnosis that it is necessary to specially consider the technique of the required manipulation. By the aid of instruments, constructed for the purpose a skilled practician may perform esophagoscopy and determine the condition of the mucous membrane. Auscultation is a method which may be employed with some degree of diagnostic satisfaction but it is rather unreliable unless the utmost care is used. By means of a stethoscope placed on the back over the course of the esophagus the abnormal sounds of deglutition may be differentiated from the normal. To accomplish this it will be necessary for the patient to take a swallow of water, or better, some thick gruel. An expert will be able to distinguish the perverted quality of the sound, its duration and the relation in time to the beginning of the swallowing act. The sound will be feeble in case of organic stricture or it may give rise to a bubbling noise. By this method it will be possible to form an opinion as to the existence of regurgitation due to obstruction.

The most important feature of an examination is the use of a bougie. This is made of flexible whalebone with an olive-shaped piece of ivory on the end. These are made in graduated sizes, and the smaller ones are to be used first. When a bougie is introduced the patient must be seated on a low chair with the head thrown back. The index finger of the operator's left hand is to be placed well back into the pharynx, the bougie then slides over the finger and easily enters the esophagus. Slight resistance may be encountered opposite the cricoid cartilage but this is easily overcome if undue haste is not used. After a small bougie has been employed larger ones should be successively introduced. Caution should always be exercised against any forcible manipulation as it is possible to perforate the esophagus, particularly when ulceration is present. This method will determine and often cure many forms of obstruction.

ESOPHAGITIS.

Acute Esophagitis.—This disease is described as an acute inflammation of the mucous membrane of the esophagus in which the submucous tissues may be invaded.

Etiology.—The causes of acute esophagitis include traumatism from the introduction of foreign bodies, thermal and chemical agents such as the swallowing of hot drinks or corrosive poisons. Extension of catarrhal inflammation from the pharynx, infectious fevers, pharyngeal diphtheria, local diseases like carcinoma and glandular or vertebral abscess are sometimes causes. The extension of thrush from the mouth is a mycotic variety found occasionally in young children.

Pathology.—The pathologic condition is essentially a catarrhal process. The mucous membrane at first is red, swollen and covered with mucous exudate. Sloughing and disintegration of tissue may follow but ordinarily the inflammation subsides by resolution. A marked granular condition may remain for a long time following the desquamation of epithelium.

Symptoms.—The *typical symptom* is a severe burning or tearing pain during the act of deglutition; it extends the entire length of the esophagus and it may be felt beneath the sternum or in the back between the shoulders. The character of the pain varies with the degree of inflammation and a sense of soreness will exist after the act of swallowing. Some patients complain of the sensation of a foreign body in the throat and movements of the head often intensify the suffering. *Dysphagia* and *regurgitation of food* are frequently experienced. The vomitus may contain blood-stained mucus. When suppuration ensues pus may be thrown up. *Thirst* is a frequent symptom and *fever,* though rarely present, may partake of the septic form when suppuration is established. The epiglottis and the arytenoid structure may suffer from the inflammatory extension, in which case dyspnea may be manifest. *Cicatricial stenosis* may follow a protracted ulceration.

Diagnosis.—The disease is diagnosed by the characteristic pain along the esophagus during the act of deglutition and during the passage of an esophageal sound. The violent ejection of liquids and the presence of mucus, pus and blood will also be confirming signs though we must carefully decide here between acute esophagitis and carcinoma of the cardiac orifice of the stomach. Inspection of the throat and laryngoscopic examination should differentiate from diseases of the pharynx and larynx. The dysphagia of pericarditis with effusion will be recognized by its characteristic signs. The expulsion of a pseudo-diphtheritic membrane must not be confused with the esophago-mycosis of children.

Prognosis.—Recovery in a short time is the usual result. The purulent and necrotic forms are serious. Cicatricial stenosis is to be feared when strong acids or alkalies have caused the disease.

Treatment.—The *feeding of the patient* is the real complication in these cases. It is often difficult to swallow the required nourishment and frequently it will be necessary to resort to the rectal enema. The inflamed esophagus must be given as much rest as possible and therefore a liquid diet is required for some time. *Milk* is the best food but at times even this may not be tolerated. *Thirst* will be relieved by pieces of ice in the mouth when water can not be swallowed without discomfort. *Pain* may demand the hypodermatic use of morphin. When the condition is due to the ingestion of a poison the proper antidote should be used if the patient is seen in the early stages. Demulcent drinks are invariably soothing.

The remedies which may be indicated are *aconite, belladonna, bryonia, rhus tox, arnica* and *arsenic* during the stage of irritation. If the troublesome nausea is not relieved by arsenic we may almost invariably obtain relief by the use of *ipecac.* Later such remedies as *arsenicum iodid, merc. corr., hepar sulphur, nitric acid, silicea* and *sulphur* may be considered. *Cantharis,* in potency, corresponds to the early symptoms more closely than any other remedy. With this we may compare *cannabis sat.* and *kali bichromicum.*

SPASM OF THE ESOPHAGUS.

Etiology.—As this term implies a spasmodic constriction of the esophagus we must refer it to those causes which are purely spasmodic. It is a neurosis and can be traced to neurasthenic or hysteric conditions. It is due to a spastic contraction of the circular muscular fibers usually in a limited portion of the esophagus. The spasm occurs during the attempt to swallow solid or possibly liquid food. Its occurrence will be noticed more with women at the early period of life and its victims are generally neurotic. The aged may be affected and hypochondriasis frequently predisposes. Chorea and epilepsy may present this as a rare symptom and psychical influences must always be inferred. Spurious hydrophobia has occasionally caused it. Reflex irritations are the source of the trouble in certain instances. It is, therefore, necessary to examine for laryngeal, gastro-intestinal or uterine causes in many cases. The gouty diathesis has been regarded as a factor.

Symptoms.—The *attacks* of esophagismus are sudden. The dysphagia is pronounced and the sense of chest constriction is distressing. Cardiac palpitation, hiccough and hysterical convulsions may attend the spasm. The seizure is, however, usually short and the patient experiences relief as the nervous manifestations subside. Liquids can generally be swallowed but solids are regurgitated with the spasm; there are exceptional instances in which liquids will temporarily fail to pass the constriction. When food is rejected it comes up suddenly and with force, which differentiates it from organic stricture. Retro-sternal pain is sometimes complained of and choking may be unexpectedly experienced.

Diagnosis.—In making a diagnosis it will not be difficult to discover the hysterical cause and the sudden and paroxysmal attack will be recognized. The physical examination by means of a bougie will afford the most satisfaction. By gradual pressure the spasm will be overcome thus permitting the bougie to pass without difficulty. The seat of obstruction is not always the same and this fact excludes organic stricture.

Prognosis.—We may give a hopeful prognosis in the majority of cases if it is possible to locate and remove the nervous cause. It may be necessary, however, to pursue the treatment for a long time.

Treatment.—The systematic and careful passage of the esophageal bougie is the best treatment. After a short period the average case will respond favorably to this. The neurasthenic or hysterical cause must be patiently treated along the lines suggested under these separate topics. Reflex irritation will demand attention and the physician must relieve the state of fear which always encourages the paroxysms. Forced feeding is often a necessity and this will often relieve the muscular spasticity.

One of the best internal remedies is *ignatia*. It covers the symptoms attending esophageal constriction and the peculiar nervous phases as well. *Chamomilla, coffea* and *pulsatilla* may be compared. *Hyoscyamus, cocculus, gelsemium, stramonium, agaricin* and *zinc phos.* should be studied in accordance with their symptomatology.

Paralysis of the Esophagus.—This is a rare disease and is the result of some nerve lesion. Post-diphtheritic paralysis is the most common cause, though it may be a sequence of bulbar paralysis, tabes and other degenerative diseases of the nervous system. *Dysphagia* is the most pronounced symptom but obstruction is not detected by the bougie. The *prognosis* is unfavorable unless the primary central disease may be corrected by treatment. When post-diphtheritic paralysis is the causative factor we may expect favorable results on the average. The *treatment* is discussed in the chapters on the causative diseases. It may be necessary to keep the patient on a liquid diet, though in some cases solid food may pass through the esophagus easier than liquids.

STRICTURE OF THE ESOPHAGUS.

Esophageal stenosis, when not a complication of organic development, is generally due to cicatricial contraction following an ulcerative process. Corrosive agents are also common causes while congenital narrowing is a rare possibility. It may be due to pressure from aneurysmal tumors, enlarged lymphatic glands or mediastinal growths, or to syphilitic, tuberculous or malignant invasion. In rare cases it has followed impaction of a foreign body.

Pathology.—While it is possible to have a stricture of any portion of the esophagus, it is located most frequently in the lower third. It is usually from one to three inches in extent but may in rare cases affect the esophagus for a greater length. The muscular walls are usually thickened and the lumen may be narrowed to a degree of complete stenosis. The mucous membrane may lie in folds and be invaded with fibrous bands or there may be a complete cicatricial degeneration.

Symptoms.—The symptoms will vary in accordance with the degree of the stenosis. A *gradually increasing dysphagia* is the most prominent indication. The patient will experience a sense of pressure at a fixed point along the esophagus, occurring at first during the effort to swallow a large bolus of food; later it will appear even when liquids alone are taken. The weakness and spasmodic contraction of the muscular coat, as well as the stenosis, will increase the difficulty in swallowing. Food accumulating in the esophagus above the stricture will often be violently ejected; this may be differentiated from stomach contents by its alkaline reaction. Dyspnea due to the pressure of the retained food on the trachea may be present. Emaciation and increasing debility will be apparent when the stricture is of long standing.

Diagnosis.—In addition to the study of the symptoms we must settle the diagnosis by means of the bougie. This will determine the seat and degree of the stenosis. Auscultation with the stethoscope may help somewhat but it is not very reliable. The differential diagnosis must be carefully made. This is necessary to avoid the possible trauma from the use of the bougie in the case of organic growths. A careful study of the history will help in this respect. The greatest danger will come from the indiscriminate use of the bougie in *aneurysmal compression.* When the narrowing is due to this cause a physical examination may detect the pulsating tumor; or the rhythmic impulse may be communicated to the free end of the bougie when it is carefully introduced down to the point of the stricture. A neurotic history will aid in diagnosing *spasm* or *paralysis* of the esophagus. *Malignant stricture* occurs past middle life and will be attended by the symptoms which characterize the peculiar growth. These are outlined under the subject of cancer of the esophagus.

Prognosis.—The outcome of the disease depends entirely upon the degree of the stenosis and the patient's ability to take nourishment. If the obstruction may be successfully dilated it is possible to palliate for some time. A fatal termination is the common result.

Treatment.—The use of the *graduated bougies* is the most feasible treatment. These should be employed daily and in some cases the treatment may be entrusted to the patient. Should an organic growth exist this is a dangerous practice though it may be necessary for temporary relief. *Rectal feeding* may be required for a time. Gastrostomy has been employed when the stricture is located near the stomach and esophagostomy has likewise been undertaken when the lesion is in the upper part of the esophagus. Careful attention to a suitable diet will always be demanded.

DILATATION OF THE ESOPHAGUS.

This may occur as a diffuse or circumscribed enlargement of the lumen of the esophagus. The former is known as a total dilatation and the latter is described as a diverticulum.

Diffuse Dilatation.—This follows as the result of stenosis when the hypertrophy of the muscular coat fails to compensate. The walls are distended by the accumulated food, until dilatation is established. The sac is spindle shaped or cylindrical, and of course is larger, the lower the seat of obstruction. Dilatation is said to occur independently of organic stenosis. Spasmodic stenosis or traumatism is the invariable cause which weakens the muscular wall.

Diverticula.—These are recognized under two heads: traction diverticula and pressure diverticula. *Traction diverticula* are the more frequent. They are small and are frequently found in children. They arise from some traction on the esophageal wall; this may come from the contraction of tissue which is attached to the esophagus by adhesive bands. Inflammatory enlargement of the bronchial glands is a common primary cause. Sometimes these diverticula are multiple. *Pressure diverticula* are quite rare and are found most frequently in men of middle age. They are usually located at the junction of the pharynx and esophagus where the muscular wall is weak. The pressure comes from within and is usually due to the distention by a large bolus of food during rapid deglutition of non-masticated food. The lodgment of a bone or any other foreign body at this point may be a traumatic cause. In these cases the muscular coat gives way, allowing the mucous coat to form a hernial protrusion.

Symptoms.—In *diffuse dilatation* the patient may complain that the food does not enter the stomach. He experiences a feeling that it lodges along the course of the esophagus and eventually regurgitation takes place. The alkaline reaction shows that this has not entered the stomach. In cases of *traction diverticula* symptoms are not easily discovered. In *pressure diverticula* dysphagia and regurgitation are prominent symptoms. As the sac is not always freely evacuated the retained food is subject to decomposition and this gives rise to a fetid breath. As the muscular wall becomes more inactive food is swallowed only by heroic effort. Complete closure may be the eventual result.

The *esophageal bougie* will be essential in making a *diagnosis.* In case of stenosis which produces a circumscribed dilatation it may, after some effort, be passed into the stomach. When there is a diverticulum it will often be diverted from the normal course and cause confusion. In such cases a bougie, bent at an angle, may help in diagnosticating a diverticulum. Inasmuch as the usual seat of this form of dilatation is opposite the cricoid cartilage we shall know at what point to make the deflection. The gradual emaciation and exhaustion of the patient will be suggestive symptoms in addition to the other findings.

Treatment.—If the trouble is caused by a stenosis we may use the methods advised under that subject to overcome it. If this treatment is successful we may help a circumscribed dilatation. Otherwise the treatment of esophageal dilatation is not undertaken with much hope. Gastrostomy is an ultimate alternative in the vain effort to do something for an incurable disease. Nourishment will be obtained with difficulty and when this is impossible the patient is a hopeless incurable. The stomach tube will be a necessary means to this end and often it is possible to prolong an unhappy existence for some time by its use.

ULCER OF THE ESOPHAGUS.

We sometimes find an ulceration of the esophagus which simulates the ulcerative condition in the stomach. It is the result of simple or follicular catarrh of the esophagus. Necrotic ulcers may appear in the esophageal vicinity of the cricoid cartilage when the patient is greatly debilitated or well advanced in years. The purulent ulceration, if extensive, may cause stenosis or permit a rupture into the trachea, posterior mediastinum or the aorta. Ulceration at the lower part of the esophagus may occur in conjunction with gastric ulceration though this is rare.

CARCINOMA OF THE ESOPHAGUS.

Cancer of the esophagus is a common affection and it is the most frequent cause of stenosis.

Etiology.—It is a primary disease and the predisposing causes incident to carcinoma in other tissues apply here. The *exciting causes* are not well understood. Heredity and age of course have some influence and the prolonged irritation of the mucous membrane is a predisposing factor. The scar of an old ulcer may be a starting point for the malignant development. It is more frequent in the male sex and therefore alcohol and the use of tobacco are reckoned as possible causes. Chronic diseases of the stomach are also supposed to contribute to malignant growths in the esophagus. Other than this we know no more about the etiology of cancers here than in any other part of the body.

Pathology.—The new growth is invariably of epithelial formation; it begins in the mucous membrane and soon involves the other esophageal structures usually forming a ring-like mass about the tube. The lower third is more often affected, though some claim the upper third as the point of most frequent invasion. It may extend into the adjacent tissues but is generally limited to the esophagus. It may be firm and fibrous, or soft and jelly-like in structure. The ulcerative process is liable to cause much disintegration of tissue, and perforation may take place into the air passages or the periesophageal tissue.

Symptoms.—*Dysphagia* gradually increases as the stenosis develops. Liquids are finally the only food that can be taken and regurgitation soon takes place. The sloughing process may later relieve the dysphagia. The *progressive emaciation* and *cachexia* of cancer is soon recognized and increasing pain, though not always present, is liable to appear. *Digestion* is impaired and vomiting develops. The *ejecta* contains blood, mucus and cancerous fragments. *Dyspnea* will develop as pressure is exerted upon the trachea. Death is the inevitable result and may follow perforation or progressive asthenia. *The cervical glands* may be enlarged and aphonia will come from any pressure upon the recurrent laryngeal nerve.

Diagnosis.—The history of the case and the progressive signs of cancerous development ought to make a diagnosis easy. The use of the esophageal bougie may reveal some cancerous tissue on the end when it is withdrawn. This is a positive sign and confirmation may be had by means of microscopic examination. *Stricture* is a suggestive sign. We may differentiate the *spasmodic stricture* by its sudden onset, the hysterical association, the freedom from pain and its disappearance under an anesthetic. In *paralysis of the esophagus* the bougie readily passes. *Syphilitic disease* will cause the greatest doubt but the history should help here. *Intrathoracic tumors,* aneurysms and the presence of foreign bodies ought to cause no uncertainty.

Prognosis.—The result is eventual death and the treatment will accomplish little more than temporary relief.

DISEASES OF THE STOMACH.

DIAGNOSTIC TECHNIQUE.

External Examination.—To correctly interpret the physical signs manifested by the stomach in disease, it is of paramount importance to have a correct knowledge of the shape, size and position of this organ in health. The accepted description is that of a pear-shaped dilatation of the alimentary canal extending from the esophagus to the duodenum, it lying mostly in the epigastrium and the left hypochondrium, about five-sixths being to the left of the median line, and one-sixth to the right. Its dimensions when distended are as follows: Between the fundus and pylorus, ten to twelve inches; between the lesser and greater curvature, four to five inches; between the anterior and posterior walls, three and one-half inches; between the cardia and pylorus, three to six inches. The larger end is directed upward and to the left, while the lower end is deflected downward and to the right.

The cardiac orifice is on the left side near the sternal attachment of the sixth or seventh costal cartilages. The pylorus is more movable, but for ordinary purposes of diagnosis may be located to the right of the median line between the tip of the xiphoid cartilage and the junction of the seventh and eighth costal cartilages. It is thus directly under the left lobe of the liver. Each of its orifices, and the general outline of the stomach, will vary to a great extent in proportion to the gastric distension. When the stomach is moderately full the fundus reaches to the left fifth interspace in the mammillary line. The lowest point, under the same degree of distention, is found in the median line one to two inches above the umbilicus in men and one to three inches in women.

Inspection.—This does not always yield satisfactory information. Excessive distention may be detected and its peristaltic activity may be discerned. Exaggerated contractions may also be observed in thin subjects; these are due either to nervous excitement of the viscus or else they may represent the stomach's unusual effort to overcome pyloric obstruction. Epigastric pulsation is sometimes a significant symptom and enlargement of the epigastric and abdominal veins may lead to diagnostic suggestions. Undue distention and marked atony each have its meaning which may help us in making a diagnosis. Morbid growths and a distended gall-bladder may sometimes be apparent on inspection.

Palpation.—This reveals much additional information. It should be practiced in a careful and painstaking manner. With the patient lying upon the back, the thighs semi-flexed, and the abdominal walls relaxed as much as possible, the hands should be placed flat upon the abdomen and careful depression of the fingers should be made over the gastric area. Later deeper palpation may be employed and with the weight of the whole hand, if necessary. In this way we may discover undue rigidity of the abdominal wall, tenderness, the consistency and the situation of neighboring organs, and the presence of a tumor.

Percussion.—With the patient still lying upon the back and with the knees flexed, we may gain more knowledge of local conditions by systematic percussion. This is best performed with the operator's fingers, though in some cases a pleximeter will give us louder and more distinctive sounds. The percussion should begin above in the thoracic area and be continued downward to the stomach; in this manner we outline the upper border of the stomach. The lower border of gastric resonance is outlined by starting the percussion in the lower part of the abdomen and continuing upward. We shall thus discover the difference in the tympanitic resonance of the stomach and the transverse colon. This, of course, will vary in pitch, according to the amount of distention. The stomach, under moderate distention, gives a low pitched tympanitic sound; when it is more fully distended it gives a higher pitch;

when it is completely distended it gives a dull sound as all vibration is then stopped. When both the stomach and intestine are under an equal degree of distention the stomach will yield a lower note as it is a larger cavity. The presence of liquids and solids in the stomach will change the sounds, so we must always be on guard to allow for such conditions.

To facilitate perfect percussion, it will be best to distend the stomach with air by means of a double bulb spray apparatus or the introduction, separately, of the two portions of a Seidlitz powder. It is necessary to compare the relative sounds of the small intestine, the transverse colon and the stomach. For a proper differentiation of colon resonance, it will aid much to distend the colon by air introduced through a tube in the rectum. As the percussion note of the small intestine is higher than that of the large intestine, it will still be difficult to appreciate a positive difference when the small intestine contains solid matter or the colon is crowded with feces. If we fill the stomach with water, a dull percussion note is heard clearly contrasting with the tympanitic note of the gas-distended colon.

Examination of the upper part of the stomach must be limited on account of the encroachment of the liver and lungs. There is, however, an area of tympanitic resonance which is fairly uniform under most conditions. This is termed *Traube's Semilunar Space.* It is outlined by Bartlett as follows: "Above, a slightly-arched line from the sixth intercostal cartilage in the parasternal line to the ninth in the midaxillary line; to the left by the anterior edge of the spleen; and below by the left costal margin. Briefly, it covers that portion of the stomach which lies in contact with the thoracic walls. Above, this arc is to be delimited from pulmonary structure, which is best done by percussion from stomach to lung." In addition to this we find "anteriorly a portion of the abdominal wall beneath which lies the anterior wall of the stomach and this area gives gastric tympanitic resonance. On the left it is bounded by the semilunar space just described. On the right it is limited by the hepatic dulness; below, and following a curved line, it extends to one or two finger breadths above the umbilicus." The area of gastric tympany may be diminished by the encroachment of neighboring organs. We may discover this in enlargements of the spleen or the left lobe of the liver, in cardiac hypertrophy and dilatation, in pericardial and in left-sided pleural effusion. It is possible for the area of gastric tympany to be increased from the following causes: cirrhosis of the liver, retraction of the lung and gastric dilatation. In dilatation and prolapse of the stomach the tympany is greatly increased downward.

Ascultatory percussion is claimed by some as an additional aid in outlining the stomach tympany. It is practiced by placing the stethoscope over the fundus and then percussing toward this point from different directions. In this way it may be possible to differentiate between gastric and hepatic tumors. *Succussion* is still of some practical diagnostic value. Two methods are recognized, the digital and the total (hippocratic). In the former the patient is placed upon the back and the operator makes rapid depressions with the finger tips over the surface from the umbilicus to the margin of the ribs on the left side. The latter method is carried out by shaking the patient, while the ear or the stethoscope is applied over the stomach. The splashing sound will show that the stomach is not empty, and if it is discovered a long time after eating, we may infer that there is some obstruction at the pylorus, or atony of the muscular walls. There will often be some confusion between the sounds in the stomach and those in the transverse colon.

Auscultation.—This offers additional information regarding the features of physical examination. It does not, however, afford the highest satisfaction in the majority of cases. *Deglutition murmurs* may be heard if the stethoscope is placed posteriorly at the left of the spinal column near the ninth or tenth rib and in front to the left of the xiphoid cartilage. The first sound is heard at the beginning of deglutition when the food passes from the pharynx into the esophagus. It may be heard all along the esophagus and it imparts sort of a hissing sound which has little diagnostic value. Six or seven seconds later we may detect the second deglutition sound, which is due to the contraction of the lower esophageal segment; this has been described as a gurgling, clucking or splashing sound which is produced as the food passes into the stomach. The absence of these sounds will cause us to suspect cardiac stenosis.

Some knowledge may be obtained regarding the size of the stomach by listening to the trickling sounds of water as it rolls down the sides of the stomach after deglutition. Rosenbach's method of blowing air through a tube into the stomach partially filled with water, gives better auscultatory evidence. In this way it is possible to detect a bubbling sound with the stethoscope. Gradually changing the depth of the tube, we may also form an idea as to the size of the stomach. Friction murmurs, when the peritoneal surfaces are roughened by inflammation, sizzling sounds, when there is excessive fermentation in the stomach, and gurgling sounds may be heard at times, but they have slight diaguostic significance. Gastroscopy and gastrodiaphany may yet yield much information when those methods of diagnostic study reach a stage of greater perfection.

Examination of Gastric Contents.—This is the most satisfactory method of determining the gastric condition. In the majority of cases it is possible to adopt this procedure. It must not be understood, however, that it is best to resort to this technique under all circumstances. There are many ininstances in which it is not advisable to use the stomach tube for this purpose. Acute gastritis, gastric ulcer and febrile diseases will contraindicate it. Severe heart disease, aortic aneurysm and advanced arterial degenerations will also forbid it. Before the contents are taken from the stomach, the patient must be given a test breakfast. Ewald's is probably the best. It consists of simply a roll and one-half a pint of water or tea without milk or sugar. In one hour from its introduction it must be taken from the stomach. Leube and Riegel's test dinner is favored by some. This consists of beef soup (400 gm.), beefsteak (200 gm.), bread (50 gm.) and water (200 c.c.). It should be removed for testing in four hours.

The gastric contents are to be withdrawn by expression or aspiration through the stomach tube, by Einhorn's stomach bucket or by the sponge method. The former is the most common method of procedure. A stomach tube with side and end openings is passed into the stomach and the outer end is retained in a glass bottle. By the aid of the patient's forced retching and firm pressure over the epigastrium it is possible to expel the gastric contents. Should failure follow this we may then resort to aspiration by means of a Politzer bag attached to the outer end of the tube; or, better than this, it would be advisable always to use a specially constructed tube with a bulb attachment. *Einhorn's stomach bucket* is a small silver vessel attached to a silken thread and with a large perforation at the top. After this has been moistened with water, the patient is instructed to swallow it. We may know that it is in the stomach when the string, on slight traction, shows the disappearance of at least sixteen inches. This bucket is allowed to remain in the stomach for five minutes, when it is to be carefully withdrawn. This causes less irritation than the expression method and, hence, in cases of suspected ulceration, it will avoid the danger of hematemesis. Unfortunately, however, it does not obtain enough of the gastric contents for any extended examination. *The sponge method* is not satisfactory, though we may get a portion of the contents in this way when the tube or bucket cannot be used. The objection to it is that most of the contents will be squeezed out before it is extracted, and that we are liable to get a mixture of the pharyngeal and esophageal secretions.

The Acids of Digestion.—Before taking up the features of chemical examination of the stomach contents, it may be well to consider the condition of the normal gastric secretions. I quote from Tyson as follows: "In healthful conditions, in ten or fifteen minutes after food ingestion the gastric contents are acid, the acidity depending on free acids or acid salts, the latter including chiefly acid phosphates of sodium and potassium introduced in various amounts with food. At this stage the free acid recognized is lactic, which is either introduced with food or is formed in the lactic acid fermentation out of carbohydrates, especially sugar. Up to thirty to forty-five minutes the lactic acid predominates, while the tests for hydrochloric acid may be negative. Then comes a stage in which traces of HCl can be demonstrated, coexisting with, it may be, lactic acid. Finally the lactic acid disappears altogether, and at the end of an hour HCl only should be present. HCl is present from the beginning, but its recognition is interfered with, partly because the first secreted immediately combines with bases until these are neutralized. Free HCl gradually increases in amount until at the acme of digestion it reaches 0.15 to 0.2 per cent after a light meal, and 0.2 to 0.33 per cent after an abundant meal.

The examination of the contents may now proceed. The *quantity* must be considered first. If the stomach is empty at the time of the test meal, the gastric contents, obtained by expression, should not be greater than 40 c.c. or less than 20 c.c. An excess means hypersecretion and a complete absence means abnormal motor activity. The *chemical reaction* should be acid, and this we may determine by means of blue litmus paper which, of course, is turned red when dipped into the gastric contents. This only establishes the fact that the contents are acid. Congo red, which is commonly employed, is changed to blue in the presence of free acids. Benzo-purpurin solution turns dark blue in hydrochloric acid and brownish black in butyric or lactic acid.

The total acidity is a much more important determination and is obtained as follows: The gastric contents are first filtered; then 10 c.c. of this are placed in a beaker and one drop of phenol-phthalein solution is added. A burette is filled with a solution of decinormal sodium hydrate which is slowly dropped into the filtrate until there is a permanent red color. Care must be exercised not to mistake the first red tinge for the permanent redness. The burette is then read and that figure is to be multiplied by 10, which gives the total acidity. The percentage may be calculated by multiplying this figure by .00365. If the gastric contents should happen to be alkaline, the burette should be filled with a decinormal solution of hydrochloric acid and then we may proceed as before. The result will give us the alkaline percentage.

Diminished acidity usually indicates diminished gastric secretion, particularly of hydrochloric acid. We observe it in organic diseases of the stomach, fevers, chlorosis, pernicious anemia and some of the chronic constitutional diseases. Chronic gastric catarrh, achylia gastrica, and cancer of the stomach show acid deficiency even to the point of complete absence. The absence of hydrochloric acid is not always significant of gastric carcinoma, unless a condition of glandular atrophy exists. Increased acidity from hydrochloric acid indicates a state of hyperchlorhydria, and this points to a condition of irritative dyspepsia, gastric ulceration or a neurosis.

Test for free hydrochloric acid.—This is made in several ways. A common method is known as *Günzburg's test.* This reagent is composed of vanillin one part, phloroglucin two parts, and alcohol thirty parts. Two or three drops of this reagent are added to an equal amount of the gastric filtrate in a porcelain dish. This is slowly evaporated over a flame and if free hydrochloric acid is present, a rose-red color appears along the edges of the dish. Blowing will hasten the reaction. This method will reveal hydrochloric acid in the proportion of 1 to 20,000 of the diluent.

Boas' resorcin test is another method for the detection of free hydrochloric acid. The reagent is composed of resorcin five parts, white sugar three parts, and dilute alcohol one hundred parts. The procedure is the same as in Günzburg's test, and a purple-red color appears along the edges of the porcelain dish when heat is applied.

Tropeolin 0.0. A saturated solution of this is placed in a porcelain dish with an equal amount of gastric filtrate and heat is added. If free acid is present, a brownish-red or deep red color will appear. Tropeolin paper (filter paper immersed for a long time in an alcoholic solution) may be used in place of the solution, but this must be prepared recently to make it reliable.

Töpfer's dimethyl-amido-azobenzol test is determined by a 5 per cent solution of this reagent. Two or three drops are added to 10 c.c. of the gastric contents and a decinormal solution of sodium hydrate is dropped into this from a burette, until a rose-red color appears. This represents the complete neutralization of hydrochloric acid. The burette is then read and the figure, multiplied by .00365 gives the percentage of free hydrochloric acid.

Uffelmann's lactic acid test.—This reagent is made by adding a drop or two of pure carbolic acid to a weak solution of ferric chlorid, which gives an amethyst-blue fluid. A few drops of the gastric filtrate are added to this and if lactic acid is present, a canary-yellow color will be detected. If fatty acids are present there will be an ashy gray reaction. The presence of phosphates may disturb this test and for that reason it is well to mix the gastric contents with double the amount of ether. The mixture is allowed to settle and the ether is decanted and evaporated almost to dryness in a water bath. To the residue about 1 c.c. of water is added and the Uffelmann test is applied as before. If now a canary-yellow color is presented, we have positive proof that lactic acid is present.

When lactic acid is discovered in the stomach contents we may know that it has been introduced by the food, or it represents a fermentation as the result of obstruction or deficient motility. If there is impairment of the motor power and lactic acid is present, while hydrochloric acid is absent, we may presume that gastric cancer exists. The simple absence of hydrochloric acid, however, does not constitute proof that there is a cancer. Boas has suggested a test meal which will obviate the presence of lactic acid from the food. He advises one or two litres of oatmeal gruel, to which is added a small portion of salt. The contents are to be obtained by expression one hour later.

Test for fatty or volatile acid.—Heat a few c.c. of the gastric filtrate to the boiling point in a test tube; place a strip of moistened blue litmus paper over this and if fatty acids are present the blue color will be changed to red.

Test for acetic acid.—When this acid is present in large quantities it will be detected by its odor. When it is present in small amounts, it is determined by neutralizing with sodium carbonate the watery residue of the etheral extract. Neutral ferric chlorid is added to this and if a blood-red color appears we may know that acetic acid is present.

Test for propeptone.—For this test equal quantities of the gastric contents and a saturated solution of common salt are mixed in a test tube. If propeptone is present a proportionate turbidity will be detected. The addition of a few drops of acetic acid will make the test more satisfactory. The cloudiness will be dissipated by heat, but it reappears on cooling. Peptone will be detected in the following manner: After filtering the mixture employed in the peptone test, it must be made strongly alkaline by the addition of sodium hydrate solution. Then a few drops of a 1 per cent solution of cupric sulphate are added. A purple or violet red reaction will appear if peptone is present.

Test for pepsin.—A disc of coagulated white of egg, 1 c.c in diameter and 1 m.m. in thickness, is placed in 5 c.c. of gastric filtrate in a test tube. This should be kept at the body temperature for several hours. If a normal amount of pepsin is present the egg albumen will be dissolved in two to six hours. *Rennet ferment* will be discovered by the following test: Three or four drops of the gastric filtrate are added to 5 c.c. of milk in a test tube. This is placed in a cup of warm water and kept at a temperature of 100° F. for ten to fifteen minutes. If there is a normal amount of the rennet ferment the milk will be curdled in that time. *Rennet zymogen* may be determined by adding a few drops of a 1 per cent solution of calcium chlorid to the above mixture, and allowing this to stand for a few minutes. If rennet zymogen is present coagulation will take place in a few minutes. These examinations will decide as to the normal conditions of the gastric juice.

Test for Erythro-dextrin, dextrin, achroödextrin and maltose.—The reagent for this test is made by the mixture of iodin 0.1, potassium iodid 0.2, and distilled water 200. When a few drops of this are added to the filtrate dextrin will give a blue color, erythro-dextrin will show a red color and achroödextrin will produce a yellowish color. Maltose will affect no change in the color. These reactions will determine the extent of the starch digestion in case of hyperacidity or increased gastric secretion.

The degree of stomach absorption.—Penzoldt's and Faber's method is accepted for this purpose. A capsule containing one and one-half grains of potassium iodid is swallowed by the patient. Great care should be exercised that none of the iodid is on the outside of the capsule. The appearance of iodin in the saliva indicates that absorption has taken place from the stomach. To prove this, strips of well dried starch paper are moistened every five minutes or oftener with the saliva and then touched with fuming nitric acid. When iodin is present in the saliva a blue reaction takes place. Normally this should occur in six to ten minutes. Delayed reaction will indicate an impaired absorption.

Test for the motor power of the stomach.——Leube's method is to wash the stomach six or seven hours after a large meal and two and one-half hours after an Ewald breakfast. If the motor power is perfect no food particles should be discovered after these washings. Ewald and Sievers have suggested the salol test. The efficiency of this is due to the fact that the acid gastric contents have no effect upon salol, but the alkaline fluids of the intestine change it into salicylic acid and phenol. Salicylic acid is absorbed and excreted by the kidneys as salicyluric acid. This is detected in the urine by the violet color produced by the addition of neutral ferric chlorid solution (10 per cent). This is applied to a drop of urine on a piece of filter paper. The patient is to take fifteen grains of salol and the urine is to be examined every half hour. Salicyluric acid should appear in the urine in forty to sixty minutes. Any delay in this indicates tardy gastric motility.

Klemperer's test with olive oil is not a pleasant method of diagnosis. Three and one-half ounces of oil are introduced into the stomach after lavage has been performed. Two hours later the stomach is aspirated and if more than 20 c.c. or 30 c.c. is left, the gastric peristalsis is considered slow. Recently the question of the stomach's motility has been settled by mechanical means. Einhorn's gastrograph and Hemmeter's deglutible elastic bag have given some encouraging statistics in this respect. The instruments, however, require the services of an expert, so that they do not bring much aid to the general practician.

The *microscopic examination* of stomach contents yields little of satisfactory import in the study of gastric diseases. The sarcinae ventriculi may be observed in conditions of health, but when they are found in large numbers they are significant of the fermentive process observed in dilatation. The Oppler-Boas bacillus is generally present in gastric carcinoma, or in association with the presence of free lactic acid.

GASTROPTOSIS.

This term is applied to the downward displacement of the stomach though, in a sense, it refers to malpositions of the stomach in general. In extreme conditions the lesser curvature may be found midway between the ensiform cartilage and the umbilicus, while the greater curvature may approach the symphysis pubis.

Etiology.—*Sex* is a factor in the etiology as gastroptosis is present much more frequently in the female because of the habit of tight lacing, and in women after repeated pregnancies. *Muscular strain* or local injury may be reckoned as causes, inasmuch as the abdominal tonicity may thus be decreased. *Displacement* of the right kidney may also drag down the liver, the stomach and part of the intestines. *Chronic constipation,* which causes the overloaded transverse colon to sag, may cause gastroptosis. Enlargement of the liver and spleen may gradually push the stomach down as these organs become hypertrophied. *Abnormal chest conditions* and *gastrectasis* will serve either to crowd the stomach below its normal level or, in dilatation, to place the greater curvature well down in the abdominal cavity. *Other chronic diseases* and congenital or acquired weakness of the supporting ligaments are contributing factors in this malposition.

Symptoms.—The symptoms are not always prominent or characteristic. *Functional disturbance* will exist in proportion to the obstruction of the pyloric exit. *Atony* of the muscular walls and impairment of the gastric secretions by perverted innervation, will lead to various symptoms significant of gastric debility. If there is extreme compression by abnormal growths or by constant tight lacing, the *capacity may be reduced* so that even a normal meal will cause painful peristaltic action. In case of dilatation with displacement, we may observe the symptoms of gastrectasis. *Gastralgic* and *colicky pains* may attend the peristaltic impairment and constipation is liable to be a bothersome complication.

Physical examination will determine the gastric displacement, and this is best accomplished by inflation, either with effervescent mixture (tartaric acid and soda bicarbonate, each one dram in separate glasses of water) or by the introduction of air through a tube. *Inspection* will show a bulging abdomen and a corresponding hollowness in the epigastric region. *Percussion* will disclose the gastric tympany below its normal location and the absence of the same in the epigastrium. When the gastric contents are retained, either on account of the gastric malposition or muscular atony, a splashing sound may be detected.

Prognosis and Treatment.—The *prognosis* is not so unfavorable as it might seem to be. Much depends upon the contributing cause. Naturally when the organic conditions of other organs are at fault, the outcome is less hopeful. Simple displacement, however, may be endured for years without marked effect. The *treatment* pertains to the removal of the cause. The diet should be that mentioned under the treatment of gastrectasis. Functional disturbances of the stomach which may arise must be carefully prescribed for. Recumbency after meals is a good habit to establish. Abdominal binders are worthy of consideration. Gastrorrhaphy may be undertaken and it is possible that surgeons may give some relief by shortening the gastrohepatic and the gastrophrenic ligaments.

GASTRECTASIS.

Dilatation of the stomach is a condition which deserves careful consideration. That organ may be enlarged in proportion to the patient's size, and a congenital enlargement does not necessarily mean a pathologic perversion. The maximum capacity of the stomach in a normal state averages 1600 c.c. or one and one-half quarts. Any increase of this capacity, which causes functional disturbance, or the failure of the stomach to contract when it is empty, permits the fermentation of retained food and this covers the definition of the subject under question.

Etiology.—The most common pathologic causes are *pyloric obstruction* and *insufficiency of the gastric muscles.* The former prevents the natural exit of the stomach contents and overworks the muscular structure. The latter diminishes the expulsive force and thereby allows the cavity to distend. Pyloric stenosis is most frequently observed. It is due to cancer of the pylorus or cicatricial contraction resulting from ulceration or corrosive poisons. Fibroid overgrowth may cause a pyloric contraction or external compression may result from organic enlargement of the liver, pancreas or gall bladder. Displacement of the right kidney, omental growths or large gallstones frequently impair the pyloric function. Simple hypertrophy of the normal tissue and congenital stenosis are rare causes. Displacement of the stomach, adhesions secondary to peritonitis, or duodenal obstructions are also to be considered as etiologic possibilities.

Atony of the muscular coats may be due to the habitual overfilling of the stomach with food and drink. This is particularly true when there are nutritional derangements following anemia, tuberculosis, carcinoma, diabetes, neurotic disturbances and other constitutional diseases. Chronic gastritis also causes muscular debility, and other influences promote the impaired innervation which develops an imperfect peristalsis. The sluggish digestion permits an abnormal accumulation of food in the stomach and this, with the distention from unnatural fermentation, favors the gastric dilatation. In some cases we may find that the muscular weakness is caused entirely by the primary involvement of the nerves which govern gastric motility. Acute dilatation is a rare occurrence, but it has been observed in connection with specific fevers which produce rapid parenchymatous degeneration of the muscular coats. It may possibly follow from the drinking of large quantities of effervescing liquids. Boas claims it may be caused by shock; Bettman reports a case due to sudden obstruction of the pylorus and duodenum; and Fagge describes it as a paralytic distention secondary to chronic catarrhal inflammation.

Pathology.—In studying the pathology we must make due allowance for the individual peculiarities in the stomach's size. We shall invariably find the intestines displaced downward while the liver, diaphragm and spleen are pushed upward. The stomach will occupy the most of the anterior abdominal cavity, its lower border being found between the umbilicus and pubis. This line will, of course, vary in accordance with the degree of dilatation. When pyloric stenosis is the cause the fundus is mostly affected and the distention will be found to the left of the median line. The muscular walls will be either hypertrophied or atrophied. The hypertrophy may represent a uniform muscular overgrowth, or it may be found mostly at the pylorus. When cancer exists there may be a diffused thickening. Atrophy is observed more frequently in those of advanced years; it often follows the hypertrophic condition and is invariably a feature of malnutrition. The mucous membrane shows the changes characteristic of chronic gastritis.

Symptoms.—In the first stages the symptoms are not very distinctive. A sense of *epigastric fulness* and other signs of indigestion are in early evidence. *Eructations* are unpleasant and persistent. The *appetite* is sometimes lessened and at other times hunger is pronounced. Thirst is usually inordinate. *Vomiting* is the most characteristic manifestation and the quantity of the ejecta is often enormous, amounting in some cases to as much as one or two gallons. The vomited matter is watery and includes remnants of previous meals; it also contains bacteria, sarcinae and yeast fungi in abundance. The *reaction* usually shows a lessened acidity, as in the majority of cases the hydrochloric acid secretion is diminished. Sometimes the acid reaction is normal or even excessive. In the latter instance it exists as the result of fermentation which produces lactic, butyric or acetic acids. Carbonic acid and hydrogen gases are also formed by the decomposition of albuminoids, and sulphurreted hydrogen may be detected. The *retention of food* and the fermentation due to hydrochloric acid deficiency pervert the natural stomach function. In some instances the stomach is never completely emptied, either by the act of vomiting or by the passage of the contents through the pylorus. As a result of this, diminished intestinal peristalsis gives rise to obstinate constipation. The *urine* is scanty, the skin is excessively dry and a progressive anemia is followed by emaciation and general debility. In *fatal cases* a marked drowsiness, the result of toxic absorption, may be noticed before the final termination. The *regurgitation* of the acid contents of the stomach is usually very distressing as the disease progresses. *Muscular cramps* may affect the calves of the legs and the flexors of the arms. *Pains,* increased after eating, are also felt in the abdomen. Tetany is sometimes observed after lavage or violent vomiting. *Cardiac palpitation* is a natural sequence as the heart is irritated by the upward pressure of the dilated stomach. The *nervous excitement* may become so extreme at times that the patient suffers with nocturnal dyspnea or distressing insomnia.

Physical Signs.—*Inspection,* though not always a valuable aid to diagnosis, may, in emaciated cases, reveal the lower level of the greater curvature and possibly the displacement of the smaller curvature. A rounded prominence may sometimes be seen just above the umbilicus when the patient is lying down and just below the umbilicus when the erect position is assumed. A corresponding depression is observed in the epigastric region. All of these outlines will be more clearly recognized if the stomach is distended with air or effervescing fluids. The peristaltic wave, from left to right, may be seen to stop suddenly at the pylorus when stenosis is the cause; reversed peristalsis may be observed in rare instances. *Palpation* serves to confirm the peristalsis of the stomach. A splashing sound is sometimes identified by the quick pressure of the fingers over the stomach or by shaking the body when the hand is on the epigastrium. *Percussion* gives the most satisfactory evidence, auscultatory percussion being especially satisfactory. The patient should be examined while standing and the percussion should be started at the edge of the ribs in the neighborhood of the parasternal line and continued downward. The note will be tympanitic until the lesser curvature is reached, when a duller sound, due to the liquid contents, is heard; this will in turn be succeeded by tympany as the bowel is approached. The area of dulness will rise when the patient lies down. When the stomach is empty we shall be able to define an increased area of tympany extending below the umbilicus. The best method is to insert a tube and gradually fill the stomach with water; as the dull sound displaces the previous tympanitic sound we shall know that dilatation exists.

Diagnosis.—The symptoms and the physical signs enumerated above must be studied carefully in order to make a diagnosis. The vomiting and the peculiar character of the vomitus are the most suggestive symptoms. It is necessary, however, to make several careful differentiations. *Intestinal dilatation* will give none of the gastric symptoms or physical signs of gastrectasis. *Gastroptosis,* when existing independently, will show, on inflation, a lower line for both the lesser and greater curvatures. *Ascites* will be diagnosed by the presence of liquid in the lowest part of the abdominal cavity which changes with the position of the patient. Palpation may reveal fluctuation. *Megalogastria,* or "big stomach," causes no obstruction to the passage of food at the pylorus. *Ovarian cyst* should not confuse after a careful abdominal and vaginal examination.

Prognosis.—Recovery in advanced chronic cases is hardly to be expected. Malignant formations at the pylorus make the prognosis absolutely unfavorable. Acute cases may respond to treatment and there is some hope in those cases due to simple muscular atony.

Treatment.—In *malignant stenosis* treatment is almost hopeless. The best that can be done under such circumstances is to nourish the patient with frequent administrations of easily digested foods in small quantities. In all cases the work of the muscular coats must be lessened as much as possible. Gastroenterostomy or complete removal of the stomach is warranted in some cases. When the *stenosis is not organic* and when the pylorus is somewhat patent, we may expect improvement from treatment. *Diet* is an important consideration. Liquid diet is best but an excess of fluids should not be used as this may encourage fermentation and distention; solids in large amount are not well borne. The prepared foods, particularly the peptones, may be used in small quantities.

Starchy, saccharine and fatty articles must be prescribed with care. The *nutrient enema* may be the only resource for temporary support. Beef juice and scraped beef may be digested with comfort. Peptonized milk and condensed broths are often endured. Alcohol and other stimulants must be prohibited. Soft boiled eggs, egg-nogs and semisolid foods are usually of great value.

Lavage is the best method of local treatment and it should be performed daily. The patient may be instructed to employ this every evening or every morning. The stomach should always be washed until it is perfectly clean. Then the patient may take the desired food. Warm water, with a slight addition of soda bicarbonate, may be sufficient; some still claim that antiseptic solutions are best. A 4 per cent solution of boracic acid or a 1 per cent solution of salicylic acid is recommended. Faradization has given good results. This may be accomplished with one electrode over the epigastrium and another over the pneumogastric in the neck. For *internal electrization* a variety of electrodes have been advised; the external electrode is placed over the epigastrium, while the other electrode is introduced into the stomach which is well filled with water. Massage has also given favorable results in forcing the contents through the pylorus and otherwise stimulating muscular contractions.

Remedies do not offer much hope in the treatment of this disease. *Hydrochloric acid* may be of some use as a digestive agent, and when this is employed we may consider pepsin as an adjuvant. *Nitro-muriatic acid* is a valuable substitute when hepatic stimulation is desired. *Strychnia* and *nux vomica* may be considered as valuable for increasing gastric peristalsis. Agents like bicarbonate of soda, charcoal and creosote, which act as anti-ferments, have been employed and are useful in some cases. Other remedies have reference to the condition of chronic gastritis which ensues and will be studied under that subject.

ACUTE CATARRHAL GASTRITIS.

This refers to an acute inflammation of the mucous membrane of the stomach. The cause is found in a nonspecific irritation from decomposing and fermenting foods. The inflammation is moderate in intensity and is attended by slight local and constitutional disturbance. Acute dyspepsia, gastric fever and simple gastritis are synonymous terms.

Etiology.—To a certain extent the cause of this disease may be found in a predisposition. Some are prone to gastric disturbance from the slightest irritation; in many cases it is an hereditary habit. Carelessness in diet, overloading the stomach, highly seasoned foods and alcoholic excess are common causes. Constitutional factors like tuberculosis, gout and rheumatism, the anemias and all diseases which encourage malnutrition, invariably predispose. Diseases of the liver, heart and lungs favor secondary congestion of the digestive tract and, therefore, may incite acute gastritis. The ingestion of over hot food or drinks, and nervous conditions which diminish the secretion of gastric juice must also be kept in mind as direct and indirect causes.

Pathology.—In the early stages the mucous membrane is red, swollen and studded with hemorrhagic extravasations. Later there is a uniform coating of mucus which is the characteristic morbid condition. Effused serum, leukocytes and desquamated epithelium are added to the gastric exudate as the disease progresses. The solitary lymph glands are frequently infiltrated and the gastric tubules may be filled with granular cells.

Symptoms.—*Loss of appetite,* nausea and epigastric pain and distress after taking food are characteristic symptoms. There is an unpleasant taste in the mouth, thirst is often pronounced, the tongue is coated heavily and the patient complains of dizziness and fulness in the head. *Vomiting* gives relief. The ejected matter contains an excess of mucus, and lactic and fatty acids will be found in the undigested food particles. The undigested food is usually the cause of vomiting, and when this is expelled the patient is invariably relieved for some time. The weakness is often profound and the nausea returns as soon as food is again taken into the stomach. *Jaundice* is present whenever the inflammation extends into the duodenum and causes any obstruction of the common duct. A sense of epigastric fulness or weight is a common symptom, unless the stomach is kept free of food, or the simplest liquid diet is employed. The *face* is pale and the extremities are cold. *Herpetic vesicles* may be noticed on the lips during the later stages of the disease, and the urine is scanty and high colored.

Fever is absent in most cases. There is a form, however, which has a distinctive febrile character. In such cases the constitutional symptoms are marked and a chill precedes the febrile action. The temperature ranges from 101° to 103° F., but does not continue long. There is an *erythematous variety* which follows the exanthematous fevers. In this form we observe little coating on the tongue and the vomitus does not contain much mucus. The marked dryness and redness of the tongue is expressive of a similar condition in the esophagus and stomach. The constitutional symptoms are often extreme and a high fever is attended by a rapid pulse.

Diagnosis.—The average case will be easily diagnosed by the foregoing characteristic symptoms. The febrile cases will offer the most doubt, as it will be difficult at first to decide between this and infectious fevers. *Typhoid* will often be suspected, but the fever of gastric catarrh will present a more rapid rise of temperature and little, if any, remission, and this disease usually disappears in a few days. The other diagnostic points in typhoid will help to differentiate as the disease progresses.

Prognosis.—The outcome is favorable in most cases. The disease does not usually last longer than three to five days.

Treatment.—When the cause is removed improvement invariably follows if a judicious diet is pursued. The vomiting is due to the undigested food and when this is ejected the stomach will generally right itself. When vomiting does not take place naturally it is best to use a *mild emetic* to encourage it. Warm water will usually accomplish this. If the bowels are constipated a mild cathartic or an enema is essential. Carbonated waters are invariably comforting to the stomach. Food may be kept from the stomach for some time, and when it is administered, milk diluted with Vichy water will be most agreeable. Light soups may then be used and a solid diet should be approached gradually. *Stomach rest* is demanded until nausea and vomiting disappear.

The *remedies* which are most frequently indicated are *ipecac,* which corrects the vomiting, *nux vomica,* which restores the normal gastric secretion and stimulates a healthy peristalsis, *arsenicum,* which is always a good tonic to a weakened or inflamed mucous membrane, and *pulsatilla,* which corresponds to many of the general and local symptoms. *Cerium oxalate* is often valuable in persistent vomiting which yields to no other remedy. *Antimonium crudum* refers to the coated tongue, and *bryonia* has similar symptoms, with thirst and gastric irritability. *Tartar emetic* and *apomorphia* will answer in protracted cases where the mucus is excessive and the vomiting and nausea persist. The febrile symptoms are generally covered by *aconite* and *belladonna.*

ACUTE TOXIC GASTRITIS.

This form of gastritis is an acute inflammation of the stomach following the ingestion of irritant or corrosive poisons. Arsenic, phosphorus, acids, caustic alkalies and corrosive sublimate are the more frequent causative agents.

Pathology.—The pathologic changes depend decidedly upon the character of the toxic irritant, the amount taken and the condition of the stomach at the time. The non-corrosive poisons create intense hyperemia, tumefaction and desquamative changes, while the corrosive substances induce necrosis of the mucous membrane and eventual involvement of the muscular coats. Alcohol and phosphorus, in addition to the acute inflammation, may lead to a fatty metamorphosis of the glandular epithelium and muscular stroma.

Symptoms.—The symptoms also vary in accordance with the character of the poison. *Intense burning pain,* tenderness on pressure and vomiting of blood or fragments of mucous membrane are prevailing symptoms of all toxic irritants. The *general symptoms* of profound prostration, cold surfaces, rapid pulse and increased respiration, with other signs of fatal collapse, and later the development of fever are invariably in evidence. Unless prompt aid is rendered or the dose of poison is small, death usually follows. After relief by an antidote, hematuria, gastric ulceration or esophageal constriction and other serious sequelae are frequent. When recovery is tardy, areas of cicatricial formation may develop and these will lead to gastric contraction and general perversion of the stomach function.

Diagnosis.—In addition to the typical symptoms we must look for signs of the toxic agent which has been swallowed.

These symptoms may often be detected by the corrosive action upon the mouth and pharynx, or even by the odor of the breath. Chemical examination of the vomitus or of the gastric contents, obtained by expulsion, will aid much in this respect.

Diphtheritic gastritis is a term applied to gastric inflammation which is due to an extension of faucial or laryngeal diphtheria. It is also the result of typhus or typhoid fever, smallpox, scarlet fever, pneumonia and similar infections. It is recorded as an occasional primary affection in children of the strumous habit.

Mycotic gastritis is a rare form of gastritis due to fungus poisons which affect the mucous membrane of the stomach. The yeast fungus, the penicillium and the sarcina, which develop as the result of gastric retention, are the most common exciting causes.

Prognosis.—This varies in accordance with the poison, the amount taken and the condition of the gastric mucous membrane. If death does not occur immediately the resultant gastritis is liable to be fatal. If an antidote is promptly employed and the stomach is relieved by active vomiting, the condition may be helped by a bland diet and the use of internal remedies.

Treatment.—The use of a *proper antidote* is of first importance. *Free vomiting* should then be encouraged; *apomorphia,* hypodermatically administered (grs. 1/10) will usually accomplish this quickly. It is usually best to wash the stomach by means of the tube, which should be used very cautiously. Demulcents and antidotes may be added to the warm water. *Mucilaginous drinks* and milk should be given after this has been done. If an alkali or an acid has been swallowed it should be antidoted before the stomach is emptied. Calcined magnesia, three ounces to a pint of water, will be serviceable when acids have been taken, while solutions of tartaric acid, lemon juice or vinegar may be employed for alkalies. It may be necessary to relieve pain by means of morphin. Ice, taken internally or applied to the epigastrium, will give much relief to subsequent symptoms. Water and milk may be given as freely as the stomach will tolerate them. Broths and a light diet may be gradually instituted. The further treatment will apply to that given under acute gastritis and ulceration of the stomach.

PHLEGMONOUS GASTRITIS.

Phlegmonous gastritis is a rare form of suppurative inflammation involving the submucous and muscular coats of the stomach. The purulent infiltration may be diffuse or circumscribed, and it occurs either idiopathically (*very* rare) or as the result of metastasis. In idiopathic cases we may find alcoholic excesses, dietetic errors and possibly traumatism as causes. Metastasis occurs as the result of pyemia, puerperal infection and exanthematous diseases. The abscesses are usually small and multiple. Peritonitis may exist as the result of gastric perforation.

Symptoms.—Severe *epigastric* and *abdominal pain* with tympany simulate peritonitis. *Vomiting,* high fever (103° or 104°), delirium, great thirst and a dry tongue are also characteristic symptoms. A frequent and irregular pulse with coma and collapse indicate a fatal termination. In moderate cases the *symptoms of typhoid* may supervene. The vomited matter may contain mucus, bile and pus. A notable feature, however, is the fact that, in many cases, the vomitus may show no sign of pus, but when it is present it is pathognomonic. A gastric tumor subsiding after the vomiting of pus is the best confirmation of the existence of an abscess.

Treatment.—The circumscribed form may demand operative measures. The medicinal treatment will accomplish little more than palliation. *Arsenicum, lachesis, iodoform, silicea, sulphur, and calcarea fluorica* may be considered from a symptomatic standpoint. *Mercurius corrosivus* and *carbolic acid* should also be thought of.

CHRONIC CATARRAL GASTRITIS.

As the term implies, this disease is characterized by a chronic catarrhal inflammation of the gastric mucous membrane. The low grade hyperemia is attended by an excessive secretion of mucus, alterations in the composition of the gastric juice and ultimate structural change in the glandular and muscular tissues. The other descriptive names are chronic gastritis and chronic catarrhal dyspepsia.

Etiology.—Continued irritation of the gastric mucous membrane is the fundamental cause of a chronic inflammation of the stomach. *Errors of diet,* irregular eating and overloading of the stomach are without doubt the earliest predisposing factors. Hot and cold drinks, the bolting of food, gastric fermentation and the immoderate eating of pastry and condiments are contributing excitants. *Alcoholic excess* and toxic agencies are directly or indirectly underlying factors, and many cases may follow functional perversions (as in neurotic cases) or the tardy recovery from the acute form. *Diseases of the liver,* heart and lungs may be the primary causes inasmuch as they may lead to a passive gastric congestion through the mechanical obstruction of the portal circulation. *Constitutional diseases,* like anemia, chlorosis, chronic tuberculosis and malaria, also favor chronic gastritis by a similar reflex circulatory perversion.

Pathology.—The anatomic changes are found mostly in the neighborhood of the pylorus. The *mucous membrane* is dark gray in color except at points where it is injected with blood; it is somewhat thickened and quite generally covered with tenacious mucus. In some places it may be slightly thinner, but at the pylorus it often presents a rough and wrinkled condition which has been defined as a gastritis polyposa. Ewald describes the morbid anatomy as a parenchymatous and interstitial inflammation. The *gland cells* may suffer erosion or granular swelling so that the tubes become changed in form or show an atypical branching. Small cysts often develop at the opening of the tubes and in the majority of cases the epithelium of the glandular structure undergoes marked degeneration. *Connective-tissue increase* between the glands may cause excessive distortion and the cells of the tubules frequently undergo mucoid transformation.

These parenchymatous and interstitial changes lead in extreme cases to an *atrophy* which impairs the secreting power of the stomach. There are two forms of this atrophy; one is characterized by the extreme thinness of the gastric coats so that the size of the stomach is increased; the other presents an enormous thickening of the coats which naturally decreases its volume. This cirrhotic condition may continue until in certain instances the gastric secretion is absolutely suspended, both on account of glandular destruction and the contraction of the organ. When the structural changes become so extensive that there is an absence of gastric secretion, the disease is termed achylia gastrica. The majority of cases do not show this extreme pathology.

Symptoms.—The symptoms which cause the patient first to complain are *loss of appetite,* even to the point of disgust for food, an unpleasant taste, a coated tongue and discomfort after eating. The *sense of fulness* or *distress* in the stomach may be attended by burning sensations with slight epigastric pain and eructations of gas. *Regurgitation* of acid or bitter food is decidedly unpleasant. All of these symptoms are aggravated as soon as food is taken into the stomach. *Nausea* and *vomiting* may take place at any time, the vomitus consisting of undigested food and a large amount of mucus. The reaction is neutral or acid; the latter being due to the presence of organic acids as the result of fermentation, rather than to the normal hydrochloric acid. All of these symptoms are liable to appear as exacerbations which simulate an acute attack. *Headache,* vertigo, disturbed sleep and depression of spirits are common complaints. The gastric distress and tenderness may be intensified until there is a general abdominal sensitiveness. Cardiac palpitation is a frequent complication and constipation is a natural result. The *urine* is scanty, the *pulse* is irregular or slow and some dyspnea may exist. *Fever* is not generally present.

The *gastric contents,* obtained after a test meal, will show a deficiency of pepsin and hydrochloric acid. As the normal acid acts as a destroyer of ferment-producing spores, the deleterious products of fermentation will be in evidence. There will also be a *deficient secretion* of gastric juice on account of the changes in the gastric tubules. The presence of *mucus,* varying in quantity and alkaline in reaction, is invariably detected. As the *atrophic condition increases* there will be a retarded peristalsis. Yeast spores and sarcinae may be mixed with a slight amount of blood. The absence of hydrochloric acid, the presence of organic acids and the quantity of mucus will depend upon the degree and chronicity of inflammation and the degenerative conditions. When *atrophy is extreme,* the contents may show no mucus and no free or combined hydrochloric acid. In that case the undigested food will be mixed with pepsin, blood, epithelium and bacteria. In addition to these special symptoms we must note the general malnutrition which pursues a prolonged case. The *anemia,* in some instances may assume the pernicious form, and death from inanition is not an uncommon result. The intestines and bile tract may become involved by direct extension, giving rise to jaundice, chronic enteritis, or a diarrhea, often alternating with constipation.

Diagnosis.—The diagnosis in the average case is not difficult when typical symptoms are in evidence. Inasmuch, however, as gastric catarrh may be a feature in ulcer, carcinoma or dilatation, it will be necessary to make careful differentiations in accordance with the symptomatology of those diseases. Ewald has made three distinguishing points in the study of chronic gastritis: (1) A simple gastritis in which hydrochloric acid is absent or diminished in the test meal, while lactic acid and fatty acids may be present. (2) A mucous gastritis in which the acidity is slight and the mucus is excessive. (3) Atrophic gastritis in which the hydrochloric acid, pepsin and curdling ferment are absent.

Prognosis.—The curability of the disease depends upon the degree of the pathologic change, the etiology, and the general condition of the patient. If the destructive lesion is pronounced, the secretion changed and the nutrition impoverished, the outcome is not hopeful. When atrophy is extreme, there is, of course, no hope for a cure. When organic diseases of the heart, lungs or liver complicate, the prognosis is always unfavorable. Tuberculosis is liable to develop during the weakened state. The majority of the cases of uncomplicated chronic gastritis should respond favorably to the symptomatic application of remedies.

Treatment.—The treatment of chronic gastritis must include, first of all, the correction of habits which induce it. The patient must be instructed as to the necessity of *regular* and *careful eating* and to accomplish this it is best to prescribe a written dietary. Rich and highly seasoned foods must be condemned and the use of stimulants absolutely forbidden. *Regular habits* of life are equally mandatory, for the dyspeptic patient is invariably irregular in all his habits. Food must be wholesome and properly masticated. Unclean or decaying teeth may be the primary factors. Another necessary factor is the use of a sufficient amount of pure water even during the meals. This is not to be used in order to bolt the food, but as an essential feature of stomach digestion. *Remote diseases* which pertain to the heart, liver, lungs or intestine must receive careful attention.

The *dietary* will require systematic study in order to meet the idiosyncrasies of each case. It is rarely necessary to resort to a strictly liquid diet, and yet this may be demanded in some cases. The patient then should be fed at least every two or three hours. Milk will answer for this purpose better than any other food. It must be prepared in accordance with the exigencies of the case. To some it is absolutely intolerable; therefore, in such instances it is best to dilute it with plain or carbonated water. Buttermilk will be tolerated by some, while diluted cream will be digested better with others. Fatty and fried food is invariably disturbing to the stomach, and when acid fermentation is excessive, starches will not be endured. Fruits are nearly always agreeable and are especially indicated when acids are deficient. Meats are not easily digested on account of the impaired gastric secretion. Thus the lighter forms of proteids and the easily digested vegetables should constitute what we may term a medium diet. The green vegetables in their season will be most serviceable, and such meats as chicken, birds and fish will complete the dietary in the treatment of this disease.

The condition of the *nervous system* must be considered in the study of diet in these cases. No dyspeptic will be able to digest food when the mental state is excited. A proper amount of nervous relaxation is necessary when the patient takes his meals, and a considerable amount of rest is required for the sake of digestion after meals. More than this, the patient should be encouraged to take a normal amount of systematic exercise. Massage may supplant this in patients suffering from exhaustion. Hygienic habits are also essential. Regular baths, preferably the cold sponge every morning, will give a healthy peripheral stimulation which cannot fail to encourage a better gastric circulation.

Lavage is one of the best agencies for the relief of this trouble. It serves not only to cleanse the stomach of its excessive amount of mucus, but acts as a stimulant in favor of a better gastric secretion and peristalsis. It should be performed every morning before breakfast by the patient, if possible. In many cases it will be best for the physician to attend to this. If there is no distress in this performance, several quarts of warm water may be passed through the stomach. A safe rule to follow is to continue the lavage until the water returns quite clear. When there is an excess of mucus, a two per cent. solution of sodium bicarbonate, or a one per cent. solution of sodium chlorid may be used. Plain water is better in some cases. If antiseptics are indicated, a two per cent. solution of resorcin or a one per cent. solution of salicylic acid may be substituted. In conjunction with this it is invariably a good principle to relieve the bowels by simple catharsis, unless that function is naturally performed. Faradism has recently become a popular method of local treatment. This is accomplished by the introduction of one electrode into the stomach and the other over the abdomen. Inasmuch as this is usually disagreeable to the patient, it is not always expedient.

Medicines which aid digestion are valuable agents. The best of these is *dilute hydrochloric acid.* As this is deficient when the catarrhal exudate is in excess, it is evident that its first usefulness will be to restore a normal reaction. Pepsin will not act except in the presence of this. Furthermore, this acid acts as an antiseptic or overcomes the tendency to fermentation as the result of acetic or lactic acid excess. It also serves to convert pepsinogen into pepsin. Five to fifteen drops are to be given in half a glass of water soon after a meal. Atrophic cases will demand the maximum dose. *Pepsin* may help in case there should be any deficiency in this agent. Inasmuch, however, as this is rarely lacking in chronic gastritis, the use of hydrochloric acid will be sufficient to restore its action. When pepsin is used, it is best to give it, in conjunction with the hydrochloric acid, in five to ten grain doses after meals. *Trypsin* or *pancreatin* may be employed to aid digestion in the small intestine. It should be given (grs. 5) after meals. Inasmuch as the stomach acidity is liable to disturb its action it should be given in keratin coated pills, or possibly with oxgall, so that its effect will take place in the intestine. *Peptenzyme,* which contains the principles of the stomach and intestinal secretions, is claimed to be of value. *Ptyalid,* which represents the salivary secretions, is another aid to gastric digestion in these cases. Many other digestive compounds have been used with success.

The *internal remedies* from a homeopathic standpoint are quite numerous, and a few important ones follow. These we may best employ in accordance with the symptomatology which relates to their physiological action. *Nux vomica* naturally appeals to us first on account of its stimulating action on the muscular fibres of the stomach. This tonic effect is shown not only upon the involuntary muscular fibres, but also in its action upon the gland secretion. Thus it stimulates gastric activity and gastric digestion at the same time. The hunger, with aversion for food, which is such a predominating symptom, is suggestive of a gastric catarrhal exudate which is liable to invade all mucous membranes. The nausea, tendency to vomit, the epigastric fulness, the waterbrash, and the general indications of a disordered stomach are well known. Many mistakes are made in not continuing this remedy long enough to get permanent benefit. In fact, one of the common errors of practice is to use too many intercurrent remedies before a well-chosen one has a chance to prove its value. *Ignatia* and *strychnia* are always to be thought of as substitutes for nux vomica when a more powerful agent is needed to correct a tardy digestion.

Arsenic and *china ars.* act as direct tonics to a debilitated and impoverished mucous membrane. More than this, they relate to a toxic condition as the result of faulty absorption following the fermenting of food and the unhealthy state due to a catarrhal exudate. The blood state in which arsenic is indicated points always to anemia, and hence it has a cachexia which is not easily misunderstood. The symptoms are defined by a loathing for food, and the malnutrition which comes from a prolonged inefficiency of a diseased mucous membrane. Thus arsenic is a deeper acting remedy than nux vomica. Mental anxiety, exhaustion, and thirst—even though water may not be tolerated—are attendant symptoms of value in making the prescription.

Cocculus is a remedy having similar symptoms to the two mentioned above. It is not often thought of in connection with gastritis. We notice the same aversion to food, but the nausea and vomiting are excited by motion. Hence we find that cocculus patients complain of a gastric disturbance when riding. These attacks of nausea may reach the extreme in which vertigo and even syncope are attendant symptoms. Violent cramps in the stomach, together with general abdominal distention, are often distressing features. *Ipecac* has a more distressing nausea, and vomiting takes place as the result of food irritation or putrefaction. Under the symptomatology of this remedy we may always look for a collapsed feeling in the stomach.

Pulsatilla relates to a slimy or greasy gastric secretion and hence there will be a marked loss of appetite, a heavily coated tongue and aversion to fats or oils, when the remedy is indicated. Furthermore, we may note the tendency to regurgitation, the pain and sense of weight when food touches the stomach. The characteristic nervous symptoms will also be present. *Carbo veg.* will be demanded when fermentation is excessive and the eructations are acid, showing that organic acids are present. *Lycopodium* has, in addition to the symptoms of carbo veg., excessive flatulence and a sense of satiety after a slight meal. *Nux moschata* has similar symptoms when drowsiness and nervous features are abundant, and *creosotum* will correspond when the fermentation and fetid eructations are associated with an extreme burning in the stomach and a cachexia which denotes physical depletion.

Euphorbium, in its action upon the digestive tract, gives rise, in its primary action, to vomiting and purging and other symptoms simulating cholera. The cerebral disturbances are quite profound, and the irritation may lead to delirium. The eructations, nausea, vomiting, burning in the stomach and the spasmodic gastric contractions are distinctive indications. Thirst for cold drinks is even more pronounced than in arsenic, and at the same time the stomach is more tolerant of cold ingesta. While this remedy is more useful in the purely gastro-intestinal disorders, it is nevertheless a valuable agent in the gastric conditions in which violently distressing symptoms are liable to appear.

Iris versicolor is such a useful remedy in my practice that it is almost impossible for me to take up the subject of digestive imperfections without referring to it. In reality, its sphere of action is mostly confined to the liver. Its solvent action upon the bile makes it our best liver remedy. In jaundice or constipation, wherein the cause is due to a congestive state of the liver, it will empty sufficient bile into the intestine to relieve both the hepatic torpidity and the intestinal incompetency. It is, therefore, of greatest service when the gastric disorders begin in the perversion of hepatic and intestinal functions. The stomach symptoms will be defined by a loss of appetite, gaseous eructations and a vomiting of sour or bitter fluids. Jaundice, constipation and hepatic pain are invariably complicating symptoms. With this we should always compare *chelidonium, chionanthus* and *carduus.*

Hydrastis may be utilized in the advanced stages of chronic gastritis. It comes into use when the glandular structure is exhausted from atrophy or prolonged low grade inflammation. The mucus discharges from the outlets of the body are profuse, showing a long-standing functional perversion of the mucous membrane. The atonic dyspepsia of old people promptly responds to its internal use. As it gives local relief to chronic ulcerations, we may use it when the degeneration of gastric tissue has reached the stage in which pyloric ulceration is well established. *Kali bichromicum* may be compared in the chronic gastritis of beer drinkers.

Argentum nitricum represents a condition of prolonged debility in which the gastric function has been impaired for a long time. It naturally suggests a stage of connective tissue increase and for that reason it applies to the old cases which do not respond promptly to other medication. Such patients are prone to vomit large quantities of yeasty fluid and the extreme distention is relieved only by violent and explosive belching of gas.

Antimonium crudum indicates a more indolent stage of gastric inflammation and is defined by the thick and dirty grayish coating of the tongue. The breath in such cases is decidedly offensive. *Antimonium tart.* covers a class of similar symptoms, but there is a greater tendency to the vomiting of large quantities of mucus. *Bismuth* may be considered under the same head, but the catarrhal gastritis is recognized by a greater amount of pain and the vomiting is also accompanied by more pain.

Bryonia is a remedy which is too frequently neglected in chronic gastritis. Headache, a dry mouth and tongue, thirst and bitter or sour eructations are always in evidence. Pain in the stomach is an ever-present symptom, but the distress is observed mostly after eating. Such patients will eject solid food while liquids and particularly water are agreeable. *Calcarea carb.* has many symptoms which simulate the action of bryonia. Food seems to remain longer in the stomach, which explains the sensation of a lump. This shows a decided impairment of gastric motility and the evidence of a general wasting of tissue is attended by pronounced cachexia. Other remedies will be suggested in accordance with their symptoms. *Abies nigra* is often used when there is a deficiency of HCl and a sensation of a lump in the stomach.

GASTRIC ULCERATION.

Ulceration of the gastric mucous membrane is not an uncommon condition in association with serious diseases of the stomach. It is often a complication of acute infections, and it is of frequent occurrence in malignant diseases of the stomach. It occurs also in the form of hemorrhagic erosions following gastric congestions which are secondary to heart, lung and liver diseases. As the term is generally employed, it refers to the simple, round and perforating ulcer which is found in the stomach, and also in the duodenum, as far as the papilla duodenalis. It is due to a localized nutritional disturbance in the mucosa and the gradual destruction of this and the underlying tissue by the hyperacid gastric juice. It is characterized by pain, vomiting and hematemesis.

Etiology.—The period of life which furnishes the most cases may be placed between thirty and forty years. It is uncommon in advanced life and with children it is very rare. *Women* are more often affected than men. *Heredity* plays some part as a cause, and those occupations which predispose to gastric compression, and carelessness in diet, may be looked upon as possible causes. *Traumatism* is an incidental etiological factor. *Capillary embolism* followed by impaired nutrition is the most frequent exciting cause. This gives opportunity to an excessively acid gastric juice to digest the mucous membrane and form an ulcer. *Acute* or *chronic gastritis* may be the preceding factors which favor this condition. It may be *secondary to anemia,* chlorosis, or amenorrhea in which there is a diminished alkalinity of the blood. For that reason we find it more frequently among females. *Nephritis,* diseases of the heart and liver, tuberculosis and syphilis, by their congestive and debilitating sequences, are prone to favor the state which makes gastric ulcer possible. *Duodenal ulcer* is less frequent and occurs more commonly with males. Virchow attributed many cases to embolism and thrombosis. Embolism of the gastric vessels is rather uncommon, but thrombosis and punctiform hemorrhage are frequent results of excessive vomiting. The *stasis* of *circulation,* caused by these occlusions, is invariably favorable to the solvent action of the highly acid gastric juice. Böttcher claims that the ulcer is due to micrococci which he found in great numbers in the vicinity of the disintegrating tissue.

Pathology.—The shape of the typical gastric ulcer is *round or oval.* It is single, though sometimes there are several which coalesce and form a large ulcer with irregular borders. The *edges* are clean cut when the ulcer is new, or again they are terraced or sloping so as to form a cone shaped depression with its broad end upward. This has given them a "punched out" appearance. The *ulcers* are mostly small, varying in size from a pea to a dime; occasionally they are found to be as large as four inches in diameter. Their *position* is usually on the posterior wall of the stomach near the lesser curvature. The *muscular coat* generally constitutes the floor of the ulcer, though the perforation may extend to the serous coat or even to an adjacent organ which, in this case, is protected by inflammatory adhesions. The healing process leaves a cicatrix which may cause pyloric stenosis or contraction and deformity of the stomach. *Perforation* is the greatest danger, and this may cause fatal peritonitis or fistulous openings into neighboring organs. The pericardium, pleurae, lungs, gallbladder, spleen, pancreas, and duodenum may be pierced in this way. *Hemorrhage* is liable to cause sudden death when a large blood vessel is eroded.

Symptoms.—*Pain* is the most characteristic and constant symptom. While it may exist as a dull, burning or tender sensation constantly felt in the epigastric area, its more typical characteristics are defined as an intense gnawing, burning or boring pain, with periodic aggravations. Its center of greatest intensity is in the epigastrium near the xiphoid cartilage and it extends directly through to the back. The pain is always aggravated soon after the introduction of food and it usually continues until the gastric digestion is completed. This would tend to prove that both the food and the secretion of the acid gastric juice were the causes of irritation. While the food excites the ulcerated part, it nevertheless gives some digestive relief by the neutralization of the acid; yet, as the pain continues for some time after the digestive process, we may conclude that the hyperacidity is the greater cause of distress. The *gastralgic paroxysms* are sometimes diffuse or intermittent and are often increased or benefited by change of position. The severity of the seizures is usually relieved by the administration of an alkali, though many times the paroxysms are only quieted by morphin. The neurasthenic state which generally attends protracted cases will give rise to various features of pain which are, to a certain extent, sympathetic. *Vomiting,* though not a constant symptom, is still a diagnostic feature. It takes place soon after eating. Nausea and eructations of acid food, generally precedes or attends the emesis. The vomitus will show an excess of hydrochloric acid. Chronic catarrhal gastritis may obscure this somewhat.

Hematemesis is the most valuable clinical sign. It may occur as a copious hemorrhage of pure red blood or when small in amount may have a coffee-ground appearance due to admixture of food and hydrochloric acid. When the ulcer is small hemorrhage may be slight, and often, particularly in case of duodenal ulceration, it will be detected in the black stools. The attending symptoms of intense anemia and marked syncope will be recognized in copious or protracted hemorrhage. A slight rise of temperature is possible at this time. *Perforation* will be announced by sudden and violent pain, abdominal rigidity and the symptoms of peritonitis and shock. *Slight hemorrhage* may take place without premonition or other subsequent symptoms. In *extreme cases* copious hemorrhage is followed by sudden death. The symptoms of the mild form may reveal nothing more than the ordinary features of dyspepsia, and in many cases gastric ulcer is only discovered at the necropsy.

Physical Signs.—*Palpation* will aid somewhat in locating tenderness and pain, and we may find that the gastralgic attack is relieved somewhat by firm pressure. When the ulcer is at the pylorus, we may in some cases feel the induration caused by the thickened tissue. If adjacent organs are adherent, a fair sized tumor may be detected. The general symptoms of anemia, debility and emaciation will be recognized during the later stages.

Diagnosis.—When hematemesis and the characteristic pain are in evidence, the diagnosis is not usually difficult. Yet when we consider that many cases offer no sign of hemorrhage, the obscurity of the disease is recognized. Pain, more or less localized, which is increased by food, and often extends through to the back, and hyperacidity are all suggestive signs. In differentiation *gastralgia* must be first considered. In this the general health does not suffer much, the chlorosis or anemia are not so pronounced, food does not distress, pressure gives positive relief and the attacks appear at longer intervals. Gastralgia is more liable to occur in neurotic individuals, and there is rarely any pain or even tenderness during the interparoxysmal state. Hematemesis is never a symptom of gastralgia, there is no sign of a pyloric tumor and hyperacidity is not so common. Gastric ulcer is simulated by *cirrhosis of the liver* when hematemesis attends the cirrhotic changes. Yet the absence of all other symptoms of ulcer should settle the diagnosis. *Gallstone colic* may resemble the paroxysms of gastric ulcer when no hemorrhage takes place. The sudden onset and the sudden cessation, the presence of jaundice and gallstones, together with other hepatic symptoms, will differentiate. *Chronic gastritis* with hematemesis may sometimes cloud the diagnosis; but in this disease hydrochloric acid is usually deficient, the vomitus contains much mucus and if blood is present, it is small in quantity. *Tabetic crises* should be differentiated by the fulgurating pains, the lost knee jerk and other positive signs of this disease. *Intercostal neuralgia* should be eliminated by the presence of painful points along the nerves and the absence of gastric symptoms.

Cancer of the stomach will cause more confusion than any other disease, in the diagnosis of gastric ulcer. The absence of hydrochloric acid in cancer is a decided contrast; still there are cases of gastric ulcer in which an attending chronic catarrhal gastritis may diminish the acid reaction. Lactic acid is invariably present in cancer and constantly absent in ulcer. In the vomitus of cancer, the particles of food and the mucus will give the bloody ejecta a coffee-ground appearance. The pain of cancer is more constant and is not particularly aggravated by eating. The presence of a tumor, the age and the cachexia should be strong presumptive evidence of cancer. *Duodenal ulcers,* according to Burwinkel, cause pain in the right hypochondriac region; vomiting is also less frequent and jaundice is more common.

Prognosis.—The prognosis depends much upon the extent of the ulcerative process. Hemorrhage and perforation constitute the gravest signs. A fair share of cases recover if an early diagnosis is accompanied by judicious treatment. Cicatricial stenosis of the pylorus, followed by gastric dilatation, may be regarded as a hopeless condition except for surgical intervention. Carcinoma frequently develops in the tissue of an indolent ulcer.

Treatment.—The principal object of treatment should be to give the stomach as much relief as possible by a *light* and *bland diet.* Broths or albuminous preparations are particularly soothing. Often it is necessary to resort to rectal feeding for a time. Milk or buttermilk is tolerated in many cases. Eggs are the best form of solid food. In marked cases it is impossible to use a solid food, though it is apparent that, aside from the irritation, food is really needed and often craved by the stomach. DaCosta reports several cases cured by an ice cream diet. A recurrence of the hemorrhage demands the return to rectal feeding and for this purpose peptonized milk is best. The next necessity of treatment is to *reduce the hyperacidity* by means of an alkali, as well as by liquid nitrogenous foods. For this purpose there is nothing better than the bicarbonate of soda. A few grains of this may be added to a pint of warm water and the patient may be allowed to sip this slowly when the stomach is empty. Otherwise it may be given in tablet form. Water alone may aid in this respect and it may be used cold, if desired, for the double purpose of diluting the acidity and for the control of hemorrhage. Calcined magnesia and other alkalies may be used.

Lavage is truly indicated, but it may be employed only in a few cases. If it is possible to resort to this method without increasing the pain or causing hemorrhage, it should be tried. It is contraindicated in severe cases.

Pain may demand *relief* by *opiates* and hemorrhage is to be controlled by the customary methods and the indicated remedies. Pieces of ice may be swallowed, and an icebag may be applied to the epigastrium. Food should be absolutely suspended for a time. *Opium* should also be used to induce quiet and lessen hemorrhage. It may be given in quarter grain doses with one grain of lead acetate. Quarter grain doses of morphin, given hypodermatically, may serve us better than opium. As a usual thing, however, our homeopathic remedies will act better and cause less irritation. Five drop doses of *adrenalin* (1-1000) administered every two or three hours, will often stop the hemorrhage. Gelatin is also advocated. Tannic acid is not used so much in modern practice, though alum solution (one teaspoonful dissolved in a glass of warm water) may be taken in moderate doses. *Ipecac, geranium, turpentine, hamamelis, secale* and *nitric acid* will usually be indicated for this purpose. The third decimal potency is about the strength which the stomach will endure without causing vomiting. *Hydrastin hydrochlorate,* in the second or third decimal potency, is a valuable remedy for hemorrhage during the chronic stage. Hypodermatic injections of *ergotin,* in two grain doses, may be employed in emergency. The subcutaneous or intravenous introduction of normal salt solution will often control a hemorrhage and at the same time overcome shock.

Remedies for general use are not numerous. *Silver nitrate,* in quarter grain doses, has been a standard remedy. We may count on equally good results from the third decimal solution in distilled water. It meets the acute condition when pain and vomiting are conspicuous symptoms. *Uranium nitrate* refers to the acidity of the stomach and degeneration of tissue near the pylorus. *Mercurius corr.* has similar symptoms with the addition of heartburn and intestinal disturbance. *Kali bichromicum* points to the loss of appetite which indicates a progressive impairment of the gastric mucous membrane. Vomiting a sour undigested fluid, with ropy, stringy mucus, with a burning pain in the stomach, is its characteristic symptom. *Hydrastis* may be compared to this when the mucus is more profuse. *Bismuth* has been a standard remedy. It relates to the distressing gastric pressure even after the slightest introduction of food. This remedy may be compared with *bryonia, pulsatilla* and *nux vomica. Atropin,* in the sixth decimal potency, is highly recommended for the gastralgic paroxysms. *Phosphorus, ferrum phos., phos. acid, calcarea phos.,* and *arsenicum,* together with the indicated tonics, may be employed when chlorosis and anemia are factors.

CANCER OF THE STOMACH.

Etiology.—The etiology of gastric cancer is clouded by the same ignorance which prevails regarding cancer elsewhere in the body. The factors relating to it may be enumerated as predisposing causes. *Age* is an almost constant factor in its development. While it may occur under thirty, it most commonly appears after forty. *Heredity,* which would seem to be a natural influence, is not a definite cause. *Sex* has some predisposing tendencies; geographical location and surroundings in general are of slight etiologic consideration. Previous diseases have contributed no definite historic data except as they favor a prolonged local irritation. With any tendency to cancer a previous gastric ulcer becomes an important factor in its development.

Pathology.—The stomach is attacked by cancer more frequently than any other organ, excepting the uterus and breast. The pylorus and the lesser curvature are the most common locations. Nearly every variety of cancer may be found. Tyson gives the following order of frequency: "(1) Cylinder-celled epithelioma most frequent at the pylorus. (2) Medullary or soft cancer, most frequent in the smaller curvature. (3) Scirrhus, at the pylorus and in the smaller curvature, causing, especially, stenosis of the pyloric orifice. (4) Colloid, diffuse infiltration with a tendency to spread to the peritoneum and adjacent organs. (5) Melanotic. (6) Squamous epithelioma, near the cardia. All the forms start from the gland cells of the mucous membrane."

The medullary forms are liable to ulcerate and develop large fungoid masses. All the varieties are prone to produce secondary new growths in adjacent organs, though the scirrhus shows the least tendency to do this. The growths are recognized as circumscribed tumors, or as diffuse infiltrations with marked thickening of the gastric tissue elements.

Gastric cancer may be a *secondary development,* particularly when the breast is primarily involved. The size, shape and position of the stomach may be decidedly changed by these new growths. When the pylorus is obstructed, the size of the cavity will be enlarged; it is usually reduced when the cancerous infiltration is excessive. Adhesions between the stomach and adjacent organs or the abdominal wall are common, and peritonitis may develop as the result of perforation.

Symptoms.—*Dyspeptic disturbances* with anorexia and eructations are early and often deceptive symptoms. These are eventually followed by *progressive debility* and *emaciation* with the peculiar *cancerous cachexia.* Persistent and rapidly increasing *pain* is next noticed. The character of this is more liable to be burning or gnawing, though at times it is lancinating. When the cardia is affected, pain occurs immediately after eating, and when the pylorus is involved, it is experienced some time after partaking of food, but it is more or less constant. *Vomiting* then becomes a fixed factor as the disease advances, and it is clearly associated with the pain. The character of the vomitus is recognized by the peculiar "coffee-ground" appearance. This occurs as the result of the oozing of blood from the ulceration of the mucosa and the action of the gastric juice.

The *chemical examination* of the gastric contents, after a test meal, reveals an absence or a marked decrease in the free and combined hydrochloric acid. Lactic acid will invariably be present. It is a rare exception when hydrochloric acid is normal or increased in amount in case of gastric cancer. The motor and secreting function is also impaired. This condition, of course, will be more apparent when the organic development is in the neighborhood of the pylorus. The Boas-Oppler bacillus is now recognized as a conclusive proof of cancer. These bacilli are nonmotile, and form straight or zigzag chains by joining end to end. In the later stages of the disease edema of the limbs may be recognized and the evidences of pernicious anemia may characterize the cachexia. The urine is liable to become scanty and show a decided reaction for indican. Febrile excitement is not an exception, and rigors, followed by profuse sweating, are sometimes observed. As the stomach becomes dilated, the vomiting will be more copious.

Physical examination usually discloses the presence of a tumor, which may be recognized by *inspection* in emaciated cases. *Palpation* reveals an irregular, hard, nodular or movable mass. When the pylorus is involved, it may drag the lower end of the stomach below its normal location and be detected in the umbilical region. At other times it may be found in the right or left hypochondrium. If it is located near the cardia, along the lesser curvature or on the posterior surface, it will not be easily found upon examination. If the infiltration is diffuse, it may not be diagnosed except for the unusual resistance and tenderness to pressure. The artificial distention of the stomach will greatly aid in finding the tumor, and it may also be detected with more certainty when the patient is changed to the knee and elbow or the standing position. *Pulsations* may sometimes be found and *percussion dulness* is a confirming sign. *Secondary new growths* very frequently exist in the liver. With hepatic enlargement jaundice will be in evidence. The mesenteric and retroperitoneal lymph glands and also the lungs may be affected, though suggestive symptoms are not always apparent. Should the peritoneum become affected, ascites will be a characteristic symptom. As the intestines become involved, constipation, diarrhea or marked indigestion will be manifest.

Diagnosis.—With the observation of the typical signs as enumerated above, it will not be difficult to make a comparatively safe diagnosis. Confirming evidence is obtained by analysis of the stomach contents and the microscopic findings. Differentiation from *gastric ulcer* will often be difficult. Ulcer, however, is more liable to occur in younger patients. The localized and paroxysmal pain which is aggravated by food, the vomiting of pure red blood, the presence of hydrochloric acid in excess, the absence of lactic acid, pallor instead of cachexia and the failure to find a tumor except in case of an extensive cicatrix, will be additional differentiating symptoms. We must keep it always in mind that ulcer is frequently the forerunner of cancer, so that its characteristic symptoms may be supplanted later by the typical signs of cancer. In *pancreatic cancer* the tumor is inaccessible, intestinal indigestion is marked and jaundice is frequently present. Diarrhea is a bothersome symptom, fats are found in the stools and sugar is detected in the urine. In *chronic gastritis* the hydrochloric acid is deficient, but pain and vomiting are not so extreme, while hemorrhage and the presence of a tumor are rare symptoms. In this disease the progressive cachexia is not present. *Tumors of the liver and spleen* will generally show an intervening tympanitic area, while *cancer of the transverse colon* will be outlined when it is distended with water or air. *Aneurysm of the abdominal aorta* presents a smooth outline and an expansile pulsation in comparison with the hard and nodular carcinomatous growth. *Duodenal cancer* will offer the most difficult differentiation. In the vomitus which occurs as the result of duodenal constriction, bile may or may not be present. If present there is an absence of HCl due to its neutralization. Otherwise HCl may be normal. Jaundice is more frequent with duodenal cancer. The cachexia of *pernicious anemia* may resemble that of cancer, but the blood picture is different. In cancer the reduction of red cells is not equal to the cachexia, while in anemia the reverse is true.

Prognosis.—The death of a patient suffering from cancer of the stomach may be expected in from six months to two years. The disease may exist in a latent form for some time before it is recognized. In such cases there may be a gradually developing asthenia which should cause us to suspect the true condition. Anders claims it will pursue a more chronic course in the emaciated subjects than in fleshy individuals.

Treatment.—It is useless to waste much space in discussing the question of treatment, inasmuch as none properly applies. It is possible, of course, to relieve pain to a certain extent and, in some cases, we shall be able to correct the excessive vomiting. *For the pain* no remedy palliates like the hypodermatic use of morphin or codein, in one-eighth or one-quarter grain doses. In advanced cases these should be used with sufficient frequency and strength to insure comparative comfort. I have formed the opinion, from my own experience, that morphin not only relieves the pain but prolongs life, or, in other words, holds the pathologic progress in restraint. Another result accomplished by this palliating agent is the decided relief from vomiting. This does not yield generally to any indicated remedy. Pieces of ice, carbonated waters or champagne may answer for this purpose temporarily. *When the stomach is dilated* on account of pyloric obstruction, the vomiting is greatly relieved by the systematic performance of lavage. *Hemorrhage,* constipation and other symptoms are to be met in accordance with instructions presented in other chapters.

The *arrangement of a diet* which will be in accordance with the digestive ability of the intestine is the only hope of prolonging life. In the majority of cases, when cancer is well established, the stomach is not able to retain or digest food. Therefore, it is necessary to prepare the food so that it demands little work on the part of the stomach and will be suitable for the intestine, providing it gets that far. The other alternative is to employ *rectal feeding.* This will be our only reliance in advanced cases. Milk may be prepared for intestinal alimentation by the addition of three to five grains of pancreatic extract and fifteen grains of sodium bicarbonate to the pint. This should be brought to a temperature of 100° F. before it is introduced into the rectum. Beef may be peptonized as follows: "Take half a pound of chopped lean meat and a quarter pound of fresh pancreas. Chop the latter and bruise in a mortar with tepid water at a temperature of 100° F. Then put both the products, together with a beaten raw egg, into a saucepan, and let it stand at a temperature of 100° F. for two hours. It is then strained for use and this amount will answer for two daily injections. No more than this should be prepared at a time."—(Tyson.) There are many preparations of predigested food on the market which often give very good results.

Those symptoms which pertain to chronic gastric catarrh, ulcer or hyperchlorhydria will receive some relief from remedies mentioned under those subjects. *Arsenic* has been employed for a long time with the hope that it was a true similimum which might bring relief. So far as records show, there is yet no proof of its ability to cure or even help. The symptoms of gastritis may be relieved by this remedy, but the pathologic process continues without any interruption. *Phosphorus, hydrastis, creosote, carbolic acid, iodin* and other suggested remedies will give no more than temporary symptomatic mitigation. Some physicians speak highly of *condurango* in gastric cancer.

Surgery has made some progress in the relief of these cases, and it is possible that by its means a cancer of the stomach may be removed and the intestine be compelled to do sufficient work to prolong life.

HEMATEMESIS.

Hematemesis is properly a symptom of some gastric disorder from which hemorrhage occurs. Gastric ulcer or cancer are the most frequent causes of this symptom. It may take place, however, as the result of traumatism within or without the stomach. Aneurysm of the arterioles, mechanical obstruction to the portal circulation, degeneration of the blood-vessels and blood diseases are also to be considered among the causes. Acute infectious diseases contribute also to the occurrence of this condition. Diseases of neighboring organs are responsible in some instances where the stomach wall is perforated. Abscess is a common factor in this respect.

Symptoms and Diagnosis.—The *typical symptom* is the vomiting of blood, which is usually dark in color, clotted and acid in reaction. We must make several differentiations to be sure of the gastric hemorrhage. *Aneurysm* of the abdominal aorta may rupture into the stomach, and it is also possible for a thoracic aneurysm to perforate the esophagus. It is also necessary to exclude hemorrhage arising from the throat, tonsils, mouth or respiratory organs. *Hemoptysis* is the most confusing sign. In this there is invariably a cough and other symptoms pointing to pulmonary disorders; the blood is raised by coughing, though it is possible for the patient to swallow the blood and then cough it up; in appearance the blood is bright red, frothy, mixed with sputum, and alkaline in reaction. In *hematemesis* we have the symptoms of gastric, splenic or hepatic disease, the vomiting of clotted blood mixed with food particles and acid in reaction. Cardiac conditions must always be kept in mind whenever hemorrhage from the mouth takes place.

Prognosis.—Hemorrhage from the stomach rarely causes death. Much depends upon the extent and persistency of the hemorrhage and the complicating diseases. At best it is a symptom of grave danger, as it is expressive of a serious underlying disease.

Treatment.—Inasmuch as the hemorrhage tends to limit itself, the best treatment is to *encourage absolute rest* by putting the patient in bed. This and other means to overcome cardiac excitement are most essential. *No food* or stimulant should be given for some time, in order to give the stomach absolute rest. *Rectal feeding* may be demanded for a time. *Ice* may be given by mouth and the icebag or coil should be applied to the epigastric area. *Morphin* may be given hypodermatically in extreme cases. This will lower the vascular tension and quiet the patient. The greatest objection to its use is the fact that, in some cases, it may cause vomiting or great nervous excitement if it does not put them to sleep. *Adrenalin chlorid* is probably our most serviceable agent to control hemorrhage. Ergot, or ergotin, by mouth or hypodermatically, are liable to produce unpleasant reactions, but may be useful in some cases. Styptics like perchlorid of iron, lead acetate, tannin, alum and turpentine present the same objections and are rarely used in modern practice except as the last resort. Normal salt solution by the mouth will often be welcomed by the patient and act as a hemostatic as well.

Internal medication is most satisfactory in the majority of cases. *Ipecac,* in the third decimal potency, not only corrects the vomiting, which is the frequent cause of the bleeding, but it acts also as a hemostatic. It will not be so useful in copious hemorrhages or when blood changes are at fault. *Cinchona, ferrum, phosphorus* and *pulsatilla* may be compared. *Hamamelis* is probably next to ipecac for its usefulness in the early stages of bleeding; its characteristic is found in the dark color of the vomited blood. The first or second potency is usually the required strength, but it may be given in the aqueous solution unless it disturbs the stomach. It is common to find some varicose veins or phlebitis in cases where this is indicated. *Trillium* and *sulphur* may be compared. Where there are traumatic causes this remedy may be compared to *arnica, hypericum* and *rhus tox.* In acute infectious cases we may be able to get good results with *aconite, belladonna, veratrum viride* or *baptisia.* When the gastric hemorrhage is associated with intestinal colic and bloody stools we may consider *agaricus, nitric acid, aloes, colocynth* and *podophyllum.*

Hydrastin hydrochlorate, in the second decimal potency, is favored by Goodno, and in severe cases he advises the hypodermatic employment of an eighth or quarter grain dose. *Turpentine* is a valuable remedy when the urinary organs are irritated or when there is excessive abdominal distention. *Erigeron* will be useful when the vomiting of blood is attended by violent retching and burning in the stomach. *Stypticin* (gr. 1-4 q. d.) is recommended for its hemostatic power. *Cantharis* may be compared with this. *Millefolium* refers to an active and profuse hemorrhage, while *crocus* relates to a hemorrhage in which dark blood clots are found in a stringy and viscid mucus. *Phosphorus, crotalus, lachesis* and *arsenicum* may be indicated when there are blood changes or predispositions similar to hemophilia. Cardiac causes must be met by such remedies as *digitalis, strophanthus, convallaria, adonis* or *glonoin.* In serious and persistent cases of hemorrhage, surgery should be resorted to as a necessary proceeding.

NEUROSES OF THE STOMACH.

GASTRALGIA.

This disease is a severe paroxysm of pain in the epigastric region without any gastric lesion or decided derangement of function. It has also been described under the terms gastrodynia, cardialgia and neuralgia of the stomach. It is evidently a neurosis of sensation.

Etiology.—The complaint is experienced most frequently by those who inherit a neuropathic tendency. Women afflicted with hysteria are particularly affected during the menstrual period or at the menopause. Excitement, worry and reflex irritations must be looked for. Food which does not easily digest may be the exciting cause. Constitutional diseases like anemia and neurasthenia are occasionally the underlying factors.

Symptoms.—Sudden and agonizing pain is experienced in the epigastric area. As this increases in severity it has a tendency to radiate into the back or around the lower ribs. The seizures may last a few minutes or an hour or more. Invariably there are moments of periodic relief, and the crisis may pass with copious eructations of gas or vomiting. A feeling of fulness or a boring and burning pain may precede the attack in some instances. Often there are such nervous symptoms as globus hystericus, reflex pains or abnormal hunger some time before the paroxysm. The patient usually bends over with the cramps or seeks relief by pressure. In many cases, however, these give no relief. Food does not generally disturb the stomach at this time and sometimes its introduction gives relief. The facial expression gives evidence of the pain and signs of syncope may follow the severe paroxysms. The subsequent prostration may continue for a long time.

Diagnosis.—The violent attacks which appear suddenly without any apparent cause, the irregular intervals and the sudden cessation of the pain are sufficiently characteristic for diagnosis. The crises which attend hyperchlorhydria, cholelithiasis and tabes must be differentiated. The pain of gastric ulcer and cancer should also be carefully compared.

Prognosis.—Death never occurs from this disease. The return to health depends upon the predisposing and exciting factors.

Treatment.—Inasmuch as gastralgia is secondary to neurotic symptoms, it is best to undertake the treatment with that fact constantly in mind. The nerve irritation which produces it must be found and removed. Thus the treatment will embrace that outlined under the subjects of neurasthenia and hysteria. If the vocation or the habits of the patient tend to produce nervous exhaustion, they must be corrected. It is obligatory to shorten the hours for business and to curtail the social demands. Regular and systematic rest are primary essentials and the physician must prescribe a definite routine in each respect. Hygienic and dietetic measures are of equal importance. The neuralgic attacks are frequently instituted by the irritation of improper food and irregular hours of eating, therefore the diet should be highly nutritious and of a character which will not tax the digestive organs.

The *treatment of the attack* will demand prompt and often heroic attention. The suffering is usually so intense that morphin (gr. 1/8-1/4) hypodermatically, is necessary. The spasm must be overcome completely. Chloroform, in combination with compound tincture of cardamon, aromatic ammonia and brandy is a valuable expedient in the majority of cases. Frequently minimum doses of any of these alone may palliate in ordinary cases. Hypnotics like chloralamid, trional, passiflora or sulfonal may answer in cases which present the typical nervous characteristics. Counter irritations and electricity may be employed successfully in some instances and pressure invariably moderates the pain to a certain extent. The treatment *during* the *interparoxysmal state* will require a well selected remedy for the relief of the irritating cause. For this purpose we must carefully study the indicated remedies. *Colocynth* seems to meet the symptoms of severe pain, with colicky cramps, but it gives little relief unless there are the associated symptoms of intestinal disturbance. *Cuprum* covers the characteristics of the seizure, but eructations and vomiting, together with a marked sense of constriction, are also recognized. *Cuprum arsenite* is the best form of this remedy. *Plumbum* gives similar symptoms, but the vomiting is liable to be more incessant, and the nausea is more pronounced and hiccough is common. The pains also are more liable to radiate to the abdomen and limbs. *Camphor* helps a burning, pressive pain in the pit of the stomach when coldness and signs of collapse appear. *Cantharis* is represented by a cutting, burning abdominal pain with the attendant symptoms of incarcerated flatus; rectal and vesical burning and tenesmus are generally present. *Terebinth* has an action similar to cantharis, but it corresponds to greater abdominal distention and refers to more renal involvement. *Platinum* relieves some cases in which the cramp-like pain is due to excessive gastric fermentation following extreme indulgence in food. *Cocculus* meets the symptoms of nausea and vomiting with cramping pain in the stomach; the symptoms are worse riding in a carriage or on ship-board.

The remedies which have a more direct relation to the peristaltic unrest as well as gastric indigestion are fairly useful. *Nux vomica* must be first considered as it pertains to peristaltic irregularity and secretory impairment. When this remedy is indicated, the gastric pressure and pain are severe and are the result of perverted muscular action and indigestion; the pain is always aggravated by food. *Strychnia* must be thought of in the same respect and particularly when these conditions are aggravated. I am inclined to use *iris versicolor* when the characteristic symptoms point more particularly to hepatic disturbance. *Pulsatilla* has some of the features of gastralgia though the distress arises from rich and undigested food. *Arsenic* is indicated when there are general symptoms of anemia and when there are pathologic signs of atrophic conditions affecting both the gastric peristalsis and glandular secretions.

For the neurasthenic cause we shall be able to find many useful remedies. *Ignatia* seems to come first as it covers so many of the mental and neurotic symptoms. The tetanic form of gastric convulsions will be accompanied by the sense of emptiness and the frequent appearance of globus hystericus. In the later stages of the disease and when the nervous symptoms are more prominent than the gastric excitement, *zincum picricum, zinc valerianate* and *cannabis indica* will be frequently indicated. There are some cases which present such active nervous symptoms that a better response will be obtained by *hyoscyamus, stramonium, opium* or *agaricin. Anacardium* exerts its greatest force upon the exaggerated mental symptoms, but when these are attended by gastralgic seizures, with the feeling of a plug or a foreign substance in the stomach, it will give good results. With this remedy we may compare *nitric acid, phos. acid, mezereum* and *abies nigra. Belladonna* must also be thought of in this disease in relation to its neurasthenic features. The suddenness of the appearance and disappearance of the paroxysms, the cutting, drawing and wrenching pain, and the hyperesthetic condition of the epigastric region are its indications. *Cimicifuga, caulophyllum, gelsemium* and *lilium tig.,* may be demanded when the fundamental fault is found in uterine disturbances. *Argentum nitricum* has a sphere of action in those cases which present a neurasthenic symptomatology as the result of degenerative diseases of the nervous system. We may state the general fact that the remedies which have the most successful application in gastralgia refer to the neurotic cause. Some of the best authorities claim that the paroxysms are due entirely to the excessive irritability of the epigastric sympathetic ganglia. This leads Buch to conclude that the descriptive term employed should be epigastralgia rather than gastralgia.

NERVOUS DYSPEPSIA.

Gastric neurasthenia is a functional disturbance of the stomach which may be traced entirely to nervous influences. The dyspeptic attacks occur somewhat regularly and without any discoverable gastric lesion. A neurotic constitution is the underlying cause and, hence, we observe the gastric manifestations in association with nervous crises and as the result of nerve irritations. Emotional and hysterical patients are most frequently affected. Overexertion, sudden excitement, anxiety, dissipation and social excess are also etiologic factors. Females with delicate nervous systems are more frequently affected than men. The life of luxury which entails too much nerve exhaustion is a prolific precursor of this form of stomach disorder. The patients rarely display any signs of gastric inability as they are invariably healthy looking. Yet underneath this good appearance we shall find evidences of nerve depletion. Temperament has much to do with the mental phases of the disease, and remote diseases may create the gastric neuroses when they create reflex irritation or destroy the cell trophism of the spinal cord. Genital and uterine irritations are the most common sources of the underlying nerve excitement. Toxemia, too, is many times the primary cause. Dietetic errors may also be regarded as provocative influences first as predispositions to neurasthenia and hypochondria and second as irritating disturbances of the gastric plexus of nerves. People inclined to weak stomachs are liable to this class of derangements whenever exhaustion or irritation of the nervous system furnishes the exciting influence.

Symptoms.—The prominent symptoms may appear in relation to the stomach, but the most characteristic symptom pertains to the neurotic cause. This must be looked for carefully in each case. In some cases anorexia is noticed, while in others we may find a voracious appetite. *Epigastric distress* after meals is a common complaint and associated with it we shall find the symptoms of regurgitation, eructations and heartburn. The *accumulation of gas* in the stomach and intestines is always a cause of complaint on the part of the patient. *Headache,* dizziness, nausea, cardiac palpitation and general discomfort are experienced from time to time. Food almost always excites these symptoms. *Vomiting* is commonly observed, but it has no relation to the taking of food or the character of the same. *Cardiac palpitation* is frequent, the patient is restless, suffers from insomnia and is generally depressed. Excitement of gastric peristalsis may be detected by palpation and, in some instances, by inspection.

Leube describes three clinical varieties of nervous dyspepsia: (1) Those in which the hydrochloric acid is present in normal amounts, which he claims is the fundamental type. (2) Those in which the hydrochloric acid is diminished. (3) Those in which the hydrochloric acid is in excess. In each of these cases the digestion is completed in the normal time except when that of starches is slightly delayed by the presence of hydrochloric acid in excess. The conclusion is thus made that nervous dyspepsia is partially a secretory as well as a sensory perversion. The hyperperistalsis would also suggest a motor complication. Chronic catarrhal gastritis may be the termination of the neurosis and hypochondriasis is not an uncommon feature.

Diagnosis.—The characteristic symptoms outlined above and the positive exclusion of other causative lesions render the diagnosis comparatively certain. The predisposing and attendant nervous features and the periodic changeableness of the symptoms should confirm the decision. The *catarrhal process* observed in chronic gastritis may at times present the symptoms of neurasthenic gastritis, and the acute attacks from toxic irritants must always be differentiated. The excess of mucus in the former, and the irritation from food in the latter will invariably aid in deciding, while the constancy of the symptoms in both will be a distinctive feature.

Prognosis.—So far as the stomach is concerned we may look for no unfavorable termination, but the nervous manifestations may never be relieved. Much depends upon the patient and the surroundings. If the neurasthenia is not cured and if the patient does not yield to treatment, the gastric perversion may continue indefinitely. When it is possible to remove the cause we may expect a complete cure.

Treatment.—The treatment of this disease is comprehended under the subjects of neurasthenia and hysteria. A *judicious* and *nourishing diet* must be insisted upon. The unfortunate tendency in these cases, as in typical nervous prostration, is to cultivate the theory of starvation. Thus tonics and remedies which stimulate the appetite may be employed with success. The change of climate and surroundings will favor the appetite and a systematic cultivation of physical exercise will develop a vigor without which the patient can not recover. *Rest* must be encouraged when the patient is overworked and diversion of occupation is necessary to overcome the natural forebodings. *Mental relaxation* will often relieve the most disturbing symptoms. The *habits* must often be corrected and stimulants of all kinds must be avoided. More than all, the physician must develop in the patient a hopeful mental attitude.

Aside from the suggestions pertaining to the neurotic phase of the disease, there are many general measures which favor a better gastric digestion. The cold sponge bath, the use of electricity and the employment of massage will invigorate the gastric function. *Lavage* will often overcome the bothersome fermentation and remove an excess of mucus. Hydrochloric acid, pepsin, pancreatin and papoid may be used when the gastric juice is insufficient or inefficient. A faulty starch digestion will be helped by the employment of a diastase after meals, and the tonic effect of *strychnia, ignatia* or *nux vomica* may be sought. *Anemia* will demand the usual treatment and nutrition must be encouraged in every possible manner. *Arsenicum* is often useful in this respect.

Other remedies are discussed in the preceding and subsequent chapters relating to diseases of the stomach and the nervous system. A careful individualization in each case will settle the true indications. *Asafetida* in the third decimal potency, corresponds to many of the gastric and abdominal symptoms which arise from nervous disorders. The pulsations felt at the pit of the stomach, the distention of the stomach and bowels, and the peristaltic excitement are relieved by this remedy. *Lycopodium* points to a flatulent condition of lithemic origin; the excessive appetite with early satiety, the pressure and heaviness in the stomach with the sense of distention which follows a tardy digestion, are frequently relieved by this. Comparison may be made with the painful, violent and burning pains of *arsenicum,* the sour vomiting, thirst and nausea of *calcarea carb.*, the vitiated appetite, hiccough and nausea of *cyclamen,* the aversion to fats, the heartburn and gnawing distress of *pulsatilla,* the unquenchable thirst and cramping pains of *natrum mur.* and the bloated sensation which appears some time after eating in *nux vomica. Argentum nitricum* will be indicated when there are violent and excessive eructations with much gastric oppression. *Anacardium* refers to a distress from incarcerated gas and all the gastric symptoms are usually relieved by eating. *Antimonium crudum* is indicative of mucus excess and easily disturbed digestion; *cuprum arsenicum* corrects the ineffectual attempts to vomit, the cramping pains which extend to the abdomen and the constriction which is reflected upward to the esophagus. *Picric acid* gives appreciable results, in association with pronounced neurasthenia, when the appetite shows decided vagaries and sour eructations are pronounced. *Bismuth,* though referring more particularly to conditions of pain, nevertheless covers many of the gastric symptoms in this disease. It is characterized by the frequent eructations and a feeling of constant distress in the stomach. The stomach will not tolerate water and all food feels like a heavy load. *Aletris* will often correct the dyspeptic symptoms when these arise reflexly from disturbance of the pelvic organs. *Crocus, helonias, cimicifuga* and *china* should also be studied. *Nux moschata* is an invaluable remedy in these conditions when, in addition to menstrual and general alimentary disturbance, brain-fag is present. *Moschus* has similar symptoms and the gastric crises partake more of the convulsive character, while spasmodic or nervous hiccough is common. There are many other remedies which are applicable to the symptomatology of nervous dyspepsia and these must be decided upon by the study of each drug.

HYPERCHLORHYDRIA.

This disease is a neurosis of secretion and is characterized by the hypersecretion of hydrochloric acid. It may occur paroxysmally in connection with great nervous excitement, and it is also an attendant condition of gastric ulcer. As a pathologic entity it must be regarded as a continuous or chronic hypersecretion which follows prolonged fasting or the introduction of irritating food in conjunction with a pronounced neurasthenic state. This variety has been known as Reichmann's disease. The etiologic factors are similar to those mentioned under nervous dyspepsia and it is most commonly found among brain workers and those exposed to nerve strain. The young are frequently predisposed and intoxicants or highly seasoned foods occasionally produce it.

Symptoms.—In the *paroxysmal forms* epigastric pain is intense and it may attend or follow the gastric function. Associate symptoms of discomfort from the ingestion of food, particularly if it is acid in character, sour eructations, heartburn, thirst and nausea are generally present. Headache and constipation are common complaints. The attacks may last for an hour or two, or they may be prolonged for a few days. Vomiting almost always terminates the paroxysm. Alkalies give relief, while acids aggravate. Large draughts of water and albuminous foods minimize the paroxysmal pain and favor digestion. Starches will not be tolerated on account of the excess of acid. Pronounced nervous symptoms are always in evidence and cardiac excitement may be marked. The tongue is red and the mouth is often sore.

In the *continuous form* there are similar symptoms, but they show no evidence of intermission. The pain is most severe when the stomach is empty, and for that reason it is noticed more at night. Food which is albuminous in character will relieve temporarily, but the distress recurs as soon as the stomach is empty. Vomiting may be copious and attended with much gaseous distention. Remnants of undigested food, excessively acid in character, may be found in the vomitus. Dilatation of the stomach is a common result in the chronic form. This may be due to the spastic closing of the pylorus under the influence of the acid irritation. It may also arise as the result of the fermenting food which is retained on account of the loss of the motor power of the stomach. In protracted cases, as glandular atrophy ensues, it is possible for the disease to be supplanted by a chronic gastritis. The patient gains little nourishment from food, and anemia and emaciation follow.

Diagnosis.—By the aid of the above symptoms we may diagnose the ordinary case, providing the neurasthenic predisposition is recognized. The analysis of the gastric contents gives most satisfactory evidence. One hour after an Ewald test meal the contents are extremely acid due to the excess of hydrochloric acid. If this condition is discovered four or six hours after a test meal, the diagnosis is confirmed. Distinction must be made when organic acids are found. Examination of the stomach contents after fasting will reveal only a small amount of gastric juice. *Gastric ulcer* has a similar hyperacidity, but food aggravates the pain and neither albuminous food nor alkalies give much relief. The nervous symptoms are not pronounced and hematemesis is a common feature.

Prognosis.—Favorable results may be expected in the majority of cases, though the continuous form is regarded as difficult to cure. If prompt treatment is instituted early in the simple forms it is possible to obviate the danger of the chronic case. Gastric ulcer and chronic gastritis are terminations which often fail to respond to any treatment.

Treatment.—The *first aim of treatment* should be to neutralize the extreme acid secretion. A *meat and milk diet* will accomplish this better than anything else; it is best to give, for a time, nothing but meat. This should be cut into small pieces or minced. Raw meat is advised by some. Objection has been made to an exclusive meat diet inasmuch as the tendency is to overstimulate the acid secretion; this, however, is not probable. Inasmuch as the stomach is liable to rebel if a fixed food is given for too long a time, it is well to vary the diet as much as possible. Eggs, fish, oysters, fowl, meats and milk will give a sufficient variety of proteid foods. Meat juices are always serviceable. Milk alone may be required in some cases and it is well to alkalinize or peptonize this. A little later the patient will tolerate a soft boiled egg and, in a careful way, it will be possible to approach a more solid diet. Before partaking of heavy meats it is well to introduce a little beef broth at the beginning of each meal. *Alkaline water* or pure spring water may be used freely with each meal, and the patient should be encouraged to drink more water between meals.

If a hearty meal is taken at night it will not be necessary to give any extra food at bed time; but if the evening meal is light, it is often advisable to give a glass of milk or a light meat sandwich upon retiring. *Starches* must generally be avoided as they cause much gaseous disturbance in a highly acid gastric juice. Stale bread or zweibach may be used with the meat and as the condition improves it will be possible to gradually add the seasonable vegetables. *Condiments* and *wines* must be discarded on account of their stimulating tendency. Eskay's albuminized food is one of the best means of nourishment in extreme cases. Food or drinks which are too hot or too cold should not be permitted. Coffee should be interdicted, though tea may be taken if it is well diluted with milk. Fats may be endured in some cases, but in the later stages of hyperchlorhydria there is danger of their producing combined acids. Sugar should invariably be avoided. In addition to the above suggestions it will be necessary always to observe regular hours for eating.

Lavage is a very useful adjuvant treatment, providing the introduction of the tube does not irritate the stomach. It is always indicated when undigested food is the cause of gastric irritation. It should be performed every morning and the water should be made alkaline by the addition of bicarbonate of soda. After this is done it will be possible for the patient to partake of a substantial meal in which albumin abounds. Alkalies, from a remedial standpoint, are serviceable. *Bicarbonate of soda* (grs. 5-10), is very beneficial and should be taken before, or immediately after eating. *Benzoate of soda* (gr. 10) may act better in some cases, while effervescent *sodium phosphate* is more agreeable in other instances. *Calcined magnesia* is still a standard remedy for this purpose as its neutralizing power is pronounced. *Lime water* will annul the acid if it is used in large doses, but the mineral waters will be more palatable to the patient.

The *constitutional treatment* needs more than casual thought. The predisposing disease may be more remote than at first seems apparent. Therefore it is always necessary to keep in mind the early features of the complaint, for these may point to a cause which is not suspected through the study of the characteristic symptoms. Chlorosis and other debilitating diseases which impoverish the blood, are frequent factors in the neurosis. Remedies like *arsenic, iron, strychnia, china* and the *calcareas* are frequently demanded and if we look carefully for the symptoms which are indicative of this status we shall find them. Should the pathologic change tend toward ulceration we must modify the treatment accordingly. If the degenerative process is more in the line of tissue transformation, then such remedies as *silver nitrate,* the *iodids, aurum chlorid, phosphorus* and the *zincs* must be thought of.

Among the remedies for the acute condition we shall get good results from the use of *atropin,* which seems to correct the excessive formation of acid. Goodno has called attention to the necessity of using it to the limit of physiological action. One to three tablets of the third decimal potency should be used before meals and at bedtime, and the dose may be gradually increased. *Robinia* has given most successful results in my practice. It presents the perfect picture of hyperchlorhydria. The intensely acid eructations, the gastric distention, the flatulence and the pain are most characteristic. *Magnesia phos.* may be used through all the different stages. It overcomes the gaseous eructations, the burning pain in the stomach, the abdominal distention and it corrects the constipation to a considerable extent. *China ars.* will embrace the indications of the two remedies which form it. It will correct the mucous membrane atonicity when this is suggested by an alternation of hyperchlorhydria and hypochlorhydria. *Hydrastis* meets a more extreme debility of the mucous and submucous tissue, there often being a catarrhal gastritis present.

Grindelia robusta is rarely used in this disease. Yet there are chronic cases in which the pneumogastric has lost its control to such an extent that gastric hyperemia is constant. This favors hyperacidity. Such cases are of pronounced neurotic type and asthma is an attending feature. This remedy, in its action upon the pneumogastric, will often be of great service. *Orexin tannate* often relieves the obstinate symptoms which attend hyperchlorhydria, but it has more particular influence on the gastric perversions which follow this disease. It has been used in two or three grain doses of the first decimal potency, before meals, when atony of the mucous membrane exists as a result of hyperacidity. Though it is most frequently used as a stomachic, its best action is found in the hyperacid conditions following neurasthenic or hysteric neuroses. Other remedies, like *ipecac, iris, bryonia* and *sulphur,* which are often indicated in gastric disturbances, may be considered in accordance with their symptomatology.

Treatment pertaining to the neurotic cause will call for remedies mentioned under the subject of neurasthenia. *Asafetida* is recognized as a standard in this respect. Its stomach symptoms are generally incidental to irritations of the generative organs and peristaltic excitement or epigastric pulsations are usually in evidence. *Conium* may be compared when there is violent vomiting of clear sour water and the eructations are also acid. *Dulcamara* simulates arsenic with its burning sensations, salivation and nausea while the nervous signs are characterized by peculiar paresthesiae and a nettlerash over the whole body. *Valerian* is a sedative for the irritable nerve centers and at the same time it relieves the spastic disturbance of the stomach which is defined by the constant disposition to vomit. *Moschus, sumbul, cannabis indica* and *avena sativa* will present similar symptoms of nervous exaggeration of the gastric symptoms. *Zinc, zinc picrate* and *picric acid, kali phos., coffea, sepia, cimicifuga* and other remedies which are frequently indicated in neurasthenia may be employed when the prominence of their cardinal symptoms is discovered.

SUBNORMAL GASTRIC SECRETIONS.

For the want of a better term we may include under this head that class of gastric disturbances in which there is a deficiency of the secretion. Subacidity denotes an abnormal decrease of hydrochloric acid while anacidity means a complete absence of the same. Illustrations of these perversions are found in such organic diseases of the stomach as carcinoma, severe gastritis, amyloid degeneration and atrophy of the gastric mucous membrane. When nervous causes are the sole factors it will be possible to exclude these organic diseases. When the hydrochloric acid and the pro-enzymes are subnormal it is a typical "hyposecretion" or "hypochylia gastrica." When these secretions are absent, the condition of anacidity is termed "achylia gastrica." The former condition is nearly always of neurotic origin, the latter is usually due to some organic structural change. Hysteria, neurasthenia and spinal diseases, like tabes, are generally the primary diseases which develop these conditions.

The *symptoms* are not always easy to differentiate. When the nervous symptoms are marked it will be possible, by analysis of the stomach contents, to determine the degree of hyposecretion. It will be best to make this examination when the nervous manifestations are at a crisis or when there is evidence of nervous shock. *Chronic gastritis* and *anemia* will furnish the pathology of this form of subacidity and, therefore, the features of these diseases may be recognized. The morbid symptoms of each form may not be discovered until the motility of the stomach is impaired and signs of intestinal irritation appear. In *achylia gastrica* nutritional impairment will be more pronounced and the stomach will be unable to digest any form of food. So long as the intestine is able to sustain life by its functions the disease may be overlooked. The abnormal putrefaction and fermentation of food will be noticed as the gastric motility is gradually lost. At the same time it may be possible to find some evidence of organic growths.

The *treatment* offers little hope. If the cause is purely neurotic and there is only derangement of secretion it is possible to obtain some relief through the removal of the nervous irritation. In these cases we may gain something from a carefully regulated diet and the correction of any intestinal weakness. Simple proteids like egg albumen, broths and meat powders may be used. Vegetables will be digested in the intestine providing the pylorus is pervious. Lavage, electricity and massage may stimulate the secretions in some cases. Hydrochloric acid, ten to twenty drops in half a glass of water, is the most rational remedy. In the typical forms of achylia gastrica there is no possibility of removing the organic cause.

NEUROSES OF MOTILITY.

Increased peristalsis of the stomach will be noticed in various forms. *Belching* and *eructations* may take place as the result of neurasthenic or hysterical causes. The air is expelled from the stomach during nervous excitement or when there is extreme gastric fermentation. *Pyrosis* is a regurgitation of the acid contents of the stomach. *Rumination* is a rare affection. It is a regurgitation of the food, which is chewed and swallowed again. *Nervous vomiting* is a neurosis which has an hysteric cause, though it may be excited reflexly by organic diseases of the liver, kidneys or uterus. It is observed also in migraine and seasickness. Though without nausea or other suggestions of gastric disturbance, it is a distressing symptom. It is not a serious condition and yields to the indicated remedy, functional rest and relief of the nervous cause. *Gastro-succorrhea* is a condition characterized by the acute appearance of a constant secretion of gastric juice giving rise to attacks of vomiting and severe pain.

Peristaltic unrest, or spasm of the stomach, is a neurotic disturbance which is often very marked in nervous dyspepsia. It is characterized by the exaggerated peristaltic movements of the stomach, the loud gurgling noises and slight amount of pain after eating. Pyloric stricture and carcinoma are frequently the causes. *Cardiospasm* is observed in nervous subjects and is manifested by a sense of cramping or constriction, with considerable tympanitic distention. The spasm is usually relieved by belching. Deglutition is often performed with difficulty and complete atresia of the cardia is a possible sequela. *Pylorospasm* is induced by the spastic action of the ring muscle of the pylorus. It may be of primary or secondary origin. Neurotic excitement, hyperacidity or organic tissue changes may cause it. The spasm is attended by pain, and the retention of food may lead to dilatation of the stomach and eventual atony of the muscular stroma.

The *treatment* of all these perversions must rely almost entirely upon the removal of the neurotic or organic causes. A strict adherence to a simple diet and relief from nervous irritation are essential. Hygienic considerations and perfect rest will also demand careful attention. Remedies which refer to the nervous and gastric symptoms may be used in accordance with the symptomatic indications.

Diminished Gastric Peristalsis.—Atony of the gastric muscles is a common result when there is any interference with the motor power. *Pyloric incompetency* is a relaxation of the pyloric muscle which has its origin in atrophic impairment as the result of a neurosis. The gastric contents pass into the duodenum before digestion is complete and a regurgitation may also take place from the duodenum into the stomach. *Insufficiency of the cardia* gives rise to eructations and regurgitations as a result of the imperfect constriction at the cardiac orifice. *Atonic dyspepsia* is a disease which is characterized by a subacute gastritis which follows insufficiency of the gastric muscles. The stomach contents, being retained longer than is natural, ferment, and cause distention, eructation of gases and an impaired appetite.

The *treatment* will correspond to that outlined under the subject of gastric dilatation. Food which has the least tendency to ferment must be selected and the quantity of liquid must not be excessive. Perfect mastication should be encouraged and all nervous excitement must be avoided. Remedies which stimulate the motor power of the stomach and a normal gastric secretion are demanded. Electricity, massage and lavage are recommended.

Hyperesthesia of the Stomach.—This is a neurosis of sensation and is allied to disorders like gastralgia. It is found in association with functional and organic diseases of the stomach, though most frequently it is a typical neurosis allied to hysteria. The stomach sensibility is so pronounced that the slightest food may cause painful sensations. The digestive process will cause an aggravation of pain, particularly if the food product is not exceedingly bland. Many times fasting is liable to produce more disturbances than the introduction of food. Hypochondriasis, neurasthenia or hysteria are invariably in attendance. Cutaneous eruptions such as urticaria and erythema may be discovered in some cases.

The treatment will comprehend the same care regarding the diet and the complicating nervous symptoms as demanded in other disturbances of this character. The nutritional features must be kept in mind both in regard to the food and the use of remedies.

Anorexia.—This is a loss of appetite which occurs generally in conjunction with organic diseases of the stomach or pronounced functional disorders. Again it may be due to a pure neurotic cause in which there is much general debility. Mental shock is sometimes the cause of its sudden appearance, and gastric hyperesthesia frequently furnishes this symptom. Debilitating diseases frequently precede it.

Hyperorexia.—Excessive appetite occurs as a gastric neurosis or it may be secondary to debilitating diseases. It may be a paroxysmal symptom (bulimia) or it may be an idiosyncrasy of habit which is illustrated by the peculiar cravings for abnormal condiments. In connection with the insatiable hunger, the patient will often complain of a burning sensation or slight pain in the stomach. Neurasthenia, chronic gastritis, tapeworm and chlorosis are frequently the causes of the symptom.

DISEASES OF THE INTESTINES.

DIAGNOSTIC TECHNIQUE.

Examination of the Feces.—Though it is an unpleasant and often unsatisfactory procedure, it is nevertheless of importance to examine the feces in order to make a complete diagnosis in intestinal diseases.

The *macroscopic* examination will give much valuable evidence regarding the shape, color and consistency of the stools. This inspection will also reveal the excess of fat, oil or mucus in the dejecta, showing thereby the imperfection of the intestinal digestion or the errors in diet, also the presence of foreign bodies, enteroliths, certain parasites, etc. The diarrheal character, the signs of constipation, the perverted color or the presence of blood will also suggest the possible cause of a diseased condition.

The *microscopic* and *chemical* examination will reveal the causes of disease and, therefore, are essential to a scientific diagnosis. The presence of blood, pus, mucus or the eggs of parasites is thus correctly established. Pathogenic bacteria, intestinal epithelium, the signs of tissue disintegration and the remnants of undigested food, when discovered in this way, will point to the organic cause or to the perversion of digestion which must be removed by treatment.

Physical Examination.—Inspection reveals many signs which are valuable. Abnormal peristalsis, unnatural prominences or contractions and the influence of respiration upon the viscera are disclosed by it. The position and mobility of a tumor may also be recognized in this way. At times it may be necessary to inflate the intestine artificially. Inspection of the rectal mucosa through a speculum is of the greatest value in determining the presence of fistulae, ulcers, hypertrophied valves, inflammations and new growths.

Palpation should be performed with the patient resting on the back and with the thighs flexed sufficiently to relax the abdominal muscles. The lateral decubitus or the knee-chest position may be required in some cases. General anesthesia is a most valuable adjuvant in this procedure. By the sense of touch we should be able to gain much knowledge regarding new growths, displacements of the intestine, over-distention, and the relation of neighboring organs. Unnatural peristalsis and increased resistance may be detected in this way. *Percussion* will disclose the presence of fluid and the encroachments from enlarged adjacent organs, new growths or abscesses. The pitch will be elevated with increased intestinal tension and lowered when there is relaxation of the bowel. It is best to inflate the large intestine in order to make proper comparisons. *Auscultation* is of slight practical value, though it may reveal some abnormal intestinal noises. One of these peculiarities is the splashing sound which may be heard in the region of the ileocecal valve in case of intestinal indigestion. The descending colon may be the seat of unnatural noises during intestinal involvement and the transverse colon will produce similar sounds which must be differentiated from those of gastric origin. Friction sounds which affect the intestine may also be detected by the stethoscope, and in a similar manner we may diagnose the excitement of peristalsis or the loss of the same in intestinal paralysis. The more complete features of diagnostic technique should be carefully studied in the larger works on that subject.

ENTEROPTOSIS.

The prolapse of the intestines from their normal position occurs independent of, or in conjunction with, the displacement of other viscera. We observe it in women as a result of tight lacing and pregnancy. It may also follow traumatism, muscular strain and the protracted use of cathartics. The colon is most frequently implicated, though the small intestine may be involved.

Symptoms.—The symptoms are not by any means well marked. Disturbance of the gastro-intestinal functions may be expected. Flatulence and other signs of intestinal indigestion are usually experienced, and the signs of enteritis may appear. The impaction of the feces will cause constipation, though we may sometimes find an alternating diarrhea. If gastroptosis and nephroptosis coexist, disturbances of these organs may be recognized. General debility, impaired nutrition and evidence of nervous derangement are often manifest.

Diagnosis.—The malposition of the intestines will be determined by physical examination. This will be greatly helped by the artificial inflation of the colon. Splashing sounds are sometimes audible and the intestine will contain more than the ordinary amount of water. When the stomach is displaced with the intestine, the pancreas may be easily palpated.

Treatment.—Careful attention to diet and the regular evacuation of the bowels will be necessary. The use of powerful cathartics should be avoided if possible. Massage and electricity may be demanded to restore the proper motor power of the stomach and intestines. Neurasthenic debility will require careful treatment for relief of nervous impairment. External support may be required to keep the intestines in their normal position. Medicinal treatment, from a symptomatic standpoint, will relieve the conditions of imperfect digestion.

ACUTE CATARRHAL ENTERITIS.

This is an acute inflammatory invasion of the mucous membrane of the intestinal tract. The inflammation is diffuse and generally pervades the larger part of the small intestine, as well as the upper part of the colon. The following terms have also been employed: Acute intestinal catarrh, acute diarrhea and acute ileo-colitis. Several types are recognized, depending upon the character of the inflammation, the location and the cause. Hence, it may be termed a mild or severe form, a duodenitis, jejunitis, ileitis or colitis, or it may be classed as a primary or secondary disease.

Etiology.—The most common cause is *intestinal irritation* and this may arise from overeating or drinking, or it may follow the introduction of acid or mineral substances. With some it may appear as the result of *exposure to cold,* or it may follow from the slightest irregularity of diet. Such patients are naturally predisposed by the most trifling digestive disturbance. The *young* and the *old* are most liable to the affliction. Sudden change of temperature—particularly the hot spells—impure drinking water and unripe food are frequent factors. The *leukomains* of fermented or decomposed food, and rich food or even the excessive eating of normal food may at times incite it. Anything which changes the character or the quantity of the intestinal secretions may lead to this affection. We may note many cases in which the excessive biliary secretion is causative, as well as deficiency of the same. Again it may be due to the *secondary intestinal hyperemia* following hepatic or cardiac diseases and traumatic causes are to be regarded as possible exciting agencies. The *contiguous inflammations* from peritonitis, or intestinal obstruction, anemia and constitutional diseases, like nephritis and tuberculosis, and all infectious diseases are possible features to be considered in the etiology. Cholera, dysentery and typhoid are the most common examples of specific infection. *Nervous influences* which tend to increase the intestinal peristalsis may be sufficient to encourage it, and often it occurs in the epidemic form. *Foreign bodies* like gallstones, enteroliths and parasites are liable to induce the disease by their constant irritation.

Pathology.—The morbid features of intestinal catarrh are not well marked. The general features of catarrhal inflammations are in evidence. Hyperemia, swelling and increased secretions will be noticed in the average case. Mucus may cover the affected portion of the membrane and the solitary glands may be the seat of pyogenic invasion; these often rupture and leave an ulceration which may extend to Peyer's patches. Hemorrhagic extravasation, associated with alterations in the mucous membrane is not an uncommon feature.

Symptoms.—The *early symptoms* may indicate nothing more than the ordinary disturbance of intestinal digestion and the onset may be either sudden or slow. Closely following the abdominal discomfort and the general malaise, a *colicky pain* will be recognized as the most prominent complaint. *Diarrhea* is the next characteristic feature and the stools, at first normal in character and color, become watery, frothy, light colored and contain mucus and epithelial debris. Flatus is distressing and the odor of the feces is offensive. Colon bacilli, yeast fungi, crystals of triple phosphates, oxalate of lime, cholesterin and particles of undigested food will also be found in the excreta. The *appetite* is impaired, thirst is extreme and a slight temperature is usually present. *Nausea* and *vomiting* will be experienced in case the stomach is simultaneously affected. Duodenitis is a frequent complication and, when this exists jaundice may be a prevailing symptom. When the disease affects the ileum and colon, mucus may be excessive in amount, and tenesmus pronounced. When the jejunum and ileum alone are affected, the pain, abdominal distention and fever will be more marked. In children delirium is often severe and the prolonged diarrhea may cause a state of collapse.

Diagnosis.—Acute catarrhal enteritis will not present any difficulty in diagnosis when the characteristic symptoms are present. If the inflammation has its seat in the duodenum, gastric complications and jaundice are determining features. If the jejunum and ileum are alone affected, diarrhea is not so prominent a symptom. If the colon is the only seat of disturbance, pain is less severe, but the diarrhea and proctitis are very annoying. It will be differentiated from *typhoid fever* by the shorter period of febrile action, the absence of the distinctive temperature curve, delirium and depression, and by the absence of the Widal reaction. *Catarrhal dysentery* has small, frequent and bloody evacuations with marked tenesmus. *Peritonitis* has more tenderness and intense pain, tympany is pronounced, the constitutional symptoms are marked and constipation is present. *Cholera* has similar but more serious symptoms and the bacteriological examination will settle the diagnosis.

Prognosis.—The uncomplicated case, occurring in a patient otherwise healthy, does not terminate unfavorably. If the disease is primary, the treatment is simple; if it is secondary to any other disease, that factor must be considered in the treatment.

Treatment.—Rest for the patient is obtained only in bed and this is a necessity when there is any febrile manifestation. Rest for the inflamed intestine is obtained only by an absolute restriction of the diet. No food should be given for twenty-four hours in the typical case, and then only the blandest form of nourishment should be prescribed. Milk and broth at stated intervals are the safest forms of diet. Milk is more acceptable to the intestine when it is boiled or given diluted with water, lime water, or soda water. Should the milk be undigested, as will be seen by the curds in the stools, farinaceous foods like arrowroot, sago, tapioca, ground rice, prepared with water and flavored with cinnamon, nutmeg or vanilla may be used. When a thick soup is found to be irritating it will be best to resort to the thinner consommé. Later this may be thickened with arrowroot, sago or tapioca. Predigested liquid foods may be serviceable. The starches and green vegetables will be endured better when the trouble is limited to the large intestine. Vegetables, fruits and fats must not be given when the upper intestine is involved. The return to solid food must be gradual and carefully watched to prevent intestinal irritation.

Catharsis, obtained by castor oil or a mild purgative or saline, may be indicated when there is an insufficiency of bile in the stools, or in case the intestine seems unable to remove impacted scybala. I am not yet converted to the indiscriminate use of calomel for this purpose, though I am free to admit that there may be occasions when a liberal dose of this, in the beginning, may expedite the cure. The high colonic flushing and often the employment of lavage may be resorted to with much benefit. Should the pain become unendurable and the diarrhea persistent, it may be expedient to use a mild form of opiate. The chlorodyne tablets, which contain a mild dose of morphin, capsicum and peppermint, are useful agents for this purpose. Morphin hypodermatically administered gives the best results in extreme cases, though opium, in the first or second decimal potencies, is often sufficient. The objection to opiates is found in the reaction which follows from locking up the intestinal secretions, and the gastric disturbance which so often ensues. Should the diarrhea show no signs of abatement, it may be permissible to use a pill containing one grain of lead acetate and a quarter brain of opium, the same as in the diarrhea or hemorrhage of typhoid fever. This may be repeated, if necessary, in three or four hours. Teaspoonful doses of chalk mixture every three hours often stop the diarrhea. In some cases it will be possible to accomplish the same result with large doses of bismuth (gr. 10-30) every three or four hours. Some help may be obtained by the local application of hot fomentations, mustard plasters or turpentine stupes. Severe pain is often relieved by the icebag. A warm flannel band or cotton compress should be worn over the abdomen until the bowel action becomes natural.

Internal astringents are not so commonly employed today, though there are times when they may be employed as adjuvants. A dram of tannic acid to a quart of water may be used in a high flushing. When the lesion is in the large intestine, small enemas of starch water, containing twenty to thirty drops of laudanum, are often efficacious. These should be repeated every three or four hours until permanent relief is obtained. When such extreme means are employed, it is best to limit the diet to simple egg albumen. To prepare this one or two eggs should be whipped up with half a pint of water and a little salt and brandy should be added. The internal use of intestinal antiseptics is frequently demanded. Methylene blue (gr. 1-2), boric acid (gr. 3-5), beta naphthol (gr. 1-4), salol (gr. 3-5) and benzosol (gr. 5-10) may be administered three or four times daily. The sulphocarbolates of zinc, soda and lime used in combination or singly, in doses of from one-sixth of a grain to five grains every two hours until the odor of the stools has disappeared certainly merits consideration. Oxgall and pancreatin compounds with small doses of nux vomica often answer this purpose and should be given soon after eating. A powder containing five grains of taka diastase and three grains of bicarbonate of soda will aid the digestion of starches and help to correct the intestinal perversion. Demulcent and mucilaginous drinks may be used freely and unfermented grape juice will be tolerated by some patients. Vichy, seltzer and other carbonated waters are usually agreeable. Sterile water and bits of ice may be relied upon to quench the thirst.

The *internal remedies* which may be indicated are quite numerous. For the febrile condition we can find nothing better than *aconite, belladonna, bryonia* or *chamomilla.* In the persistent fevers, with signs of infection, *baptisia, rhus tox, echinacea* or *gelsemium* may be demanded. For the symptom of pain *cuprum ars.* is most frequently indicated as the distress assumes a convulsive form and the cold and clammy perspiration simulates the choleraic character. *Colocynth* is naturally suggested when the paroxysmal colicky pains predominate, which are relieved by pressure. *Cantharis* is indicated by the burning dysenteric stools which resemble "scrapings of the intestines"; tenesmus is pronounced and there is a tendency to bloody discharges. *Mercurius corosivus* may be compared with this remedy. *Arsenic* is always to be considered as the debility, nervous irritation and gastric disturbance are frequently present. *Croton tiglium* may be required temporarily to control the copious watery stools; and *camphor* meets the symptoms of cramps, coldness, collapse, involuntary movements and weakness which are suggestive of cholera. *Aloes* corrects the abdominal distention which attends the enteritis, but its special action is upon the lower intestine when sphincteric incontinence is extreme. *Terebinth* refers to a tympanites in which infection is a cause, and hemorrhage from the bowels is somewhat common. *Lycopodium* controls a bloated feeling in the abdomen with which there are associated symptoms of gastric and hepatic disturbance. *Carbolic acid,* though a dangerous remedy to tamper with, may be used in potency for the relief of a diarrhea in which the putrid discharges simulate those of cantharis, and blood is more abundant.

When the disturbances are in the upper intestine we shall obtain relief from such remedies as *ipecac, podophyllum, china sulph., iris* and *chionanthus.* The last named remedy will often correct an obstinate diarrhea when used in half dram doses of the tincture. *Magnesia phos., sepia, silicea, carbo veg.,* and similar remedies will correct the abdominal distention when gastric fermentation and imperfect assimilation are the characteristic symptoms. *Veratrum alb., mercurius* and *colchicum* are indicated when there is sudden collapse of the vital powers, a low grade catarrhal inflammation, or when the symptomatology is dependent on a lithemic state. *Gambogia* is a remedy which has a decided action upon both the stomach and the intestine; it is characterized by the rumbling of gas in the intestine, the sudden and forcible ejection of watery stools and marked tenderness in the abdominal region. *Sulphur* must always be remembered for its peculiar early morning diarrhea and its other typical symptoms which appear in delicate subjects. *Phos. acid, phosphorus, cinchona, pulsatilla* and *nux vomica* have each a special individuality, and, if used in accordance with their indications, will not only check the diarrhea, but cure the disease.

CHRONIC CATARRHAL ENTERITIS.

This is of rare occurrence as a primary disease inasmuch as it is usually secondary to prolonged or repeated attacks of the acute form. The inflammatory invasion affects a greater part of the large and small intestine. Diseases which favor a passive congestion of the intestinal tract are liable to develop this form of enteritis. Therefore, the cause may originate in perversions of the heart and liver or it may be due to a defective nutrition arising from such exhausting diseases as anemia, tuberculosis or Bright's disease. Dysentery is also to be mentioned in this respect.

Pathology.—The pathologic findings in the average case represent the residual conditions of acute catarrh. Ulcerative changes, extravasations and pigmentation, thickening of the coats, and contractions from partially healed ulcers are frequently found in the lower part of the ileum and colon. The mucous membrane is covered with a muco-purulent exudate. Atrophy may invade the membrane to a considerable extent when the disease is prolonged for any length of time. Pressure ulceration or adhesions may arise as encroachment occurs secondary to cancer or tuberculosis of the peritoneum.

Symptoms.—*Chronic diarrhea* is the distinctive symptom; it may alternate with constipation, particularly when the lesion is confined to the lower intestine. *Constipation* may be the prevailing sign when atrophic changes invade the glandular and muscular structures. In the typical case the *stools are watery* or semisolid with a brownish-yellow color and a decidedly offensive odor. Undigested food is not infrequently seen, but the *presence of mucus* is the most conspicuous manifestation. This appears in flakes mixed with the feces or it may be in such large proportions that it covers the dejecta or gives the appearance of a jelly-like mass. Sometimes mucus is the only thing observed in a small evacuation. The movements seem to be more frequent during the morning hours, with an improvement during the afternoon. *Blood in the stools* is a common occurrence as the result of ulceration. When it exudes from the rectum, it is more liable to be in the form of coagula, but when it comes from a higher part of the intestine, it has a coffee-ground or tarry appearance. This is liable to occur as the result of mechanical obstruction to the venous return when the heart, liver or lungs are affected. Pus in the stools is not uncommon. It is the result of abscess formations, proctitis or a rectal fistula.

Pain, though often present, is not generally a persistent or bothersome symptom. It is more liable to be connected with a gastritis, but is not an uncommon feature of the gaseous distention in the intestine. The constitutional phases become more noticeable as the disease progresses. The nutritional disturbances will be recognized by the progressive emaciation and attendant exhaustion. When the atrophic process becomes marked the case may simulate a pernicious anemia.

Diagnosis.—The disease is recognized principally by the diarrhea and the character of the stools. In this way it is possible to determine whether the lesion represents a simple catarrhal inflammation or a structural change in which ulceration is a common factor. The *discharges* which typify the catarrhal form show an excess of mucus. The bloody and purulent passages are suggestive of ulceration, while the clay-colored stools and the undigested fats in the feces prove that the upper intestine is the seat of disturbance. The *pain* is localized near the umbilicus when the small intestine is involved and the digestive perversion is quite pronounced. When the large intestine is affected the pain is along the line of the colon and rather severe in character, but the digestive disturbance is not so pronounced. *Amyloid degeneration* has some similar symptoms, but it shows a primary affection by the enlargement of some other organ as well as the appearance of a lardaceous anemia.

Prognosis.—It is a most obstinate disease to treat and the prognosis as to a cure should be guarded. Uncomplicated cases are more tractable. If the disease shows a disposition to respond to remedies and diet, it will do so inside of ten days. The persistent diarrhea, with fever and emaciation attending, is an unfavorable symptom. Previous debility, from any cause, also favors a protracted course. Organic diseases of the heart, lungs or kidneys will make the prognosis unfavorable. Extensive ulceration may lead to incurable cicatricial contractions, while the intestinal perforation and peritonitis are possible sequelae.

Treatment.—Much that has been said under the treatment in the acute form may be applied here. *Rest* and relief from nervous excitement are important requirements. Associate organic diseases must be studied carefully. The *diet* must not be irritating, nor should the food produce much waste. Peptonized milk and simple albuminous foods will be of great service. Koumiss, tender meats, beef peptonoids, etc., should always be tried. Fruits and vegetables must be generally avoided and, should there be a gouty factor, the nitrogenous foods must be limited. Stale bread and well cooked cereals may be used when the diarrhea is not pronounced. It is best, however, to watch the diet carefully until solid stools show a return to normal conditions. *Daily irrigation of the intestine* with pure hot water or salt solution is a valuable adjuvant. Solutions of salicylic acid, 2 per cent, boric acid, 1 per cent, or a 1 per cent solution of both salicylic and boric acids, or a solution of tannic acid, 1 per cent, are advised by many experienced practicians. Corrosive sublimate, 1 to 15,000, may be used in extreme cases, but it is liable to be too irritating. Demulcents may be alternated with these solutions. *When ulceration exists,* nitrate of silver and sulphate of copper, in tenth grain doses, three times daily, may be cautiously employed. The acetate of lead alone, in one grain doses, or in combination with quarter-grain doses of opium, may be demanded when the diarrhea is obstinate and weakening. The *intestinal antiseptics* mentioned under the acute form are worthy of consideration. Bismuth subgallate, betanaphthol, bismuth and sulphocarbolate of soda in five to ten grain doses are also recommended.

The remedies which have been employed successfully are quite numerous. Many of those which are indicated in the acute form are serviceable. *Arsenicum* is naturally thought of first, and this may be used in combination with *cuprum, china* or the *iodids. Mercurius corrosivus* is indicated when the colon and rectum are involved, the symptom of tenesmus being pronounced. *Sulphur* pertains to the more chronic case in which the catarrhal discharge is of long standing. *Natrum sulph.* and *nitric acid,* in fairly high potencies, may give better results under similar conditions. *Methylene blue,* in one grain doses, will often stop the mucous discharge and overcome the intestinal irritation from putrefactive causes. *Argentum nitricum* gives as good service as in the acute form, but *aloes* will act better when the diarrhea is irritating to the rectum and flatus is constant.

In my experience *dioscorea* is one of the best remedies, and I often use it in combination with *camphor* and *chamomilla,* which have a similar action upon the intestine. It refers to griping and cutting pains in the abdomen with rectal involvement and much attending exhaustion. *Esculus, berberis* and *podophyllum* may be thought of in this connection. *Opium* in potency and *alumen* are of some use when peristalsis of the lower bowel is impaired and the stools are marble-like.

CHOLERA MORBUS.

The other terms for this disease are cholera nostras and sporadic cholera. It is an acute gastro-intestinal catarrh characterized by violent vomiting, purging and painful colic. The attack is usually self-limited.

Etiology.—It occurs most frequently during the summer months when there is a sudden change of weather and is also common to the tropical countries. Young adults and children are mostly affected; it is, therefore, confused with cholera infantum. Nervous disturbances are often the apparent cause. It has a close similarity to the true cholera, though no definite bacillus has yet been discovered. The Finkler and Prior bacillus, which resembles the comma bacillus, has been found in the discharges. The disease is evidently due to a variety of toxins resulting from decomposed food.

Pathology.—The morbid anatomy resembles that of catarrhal enteritis and undigested food products are generally discovered. Fatal cases will reveal many of the pathologic features of true cholera.

Symptoms.—The seizures appear suddenly with pain or oppression in the epigastrium. Severe gastro-intestinal cramps, vomiting and purging immediately follow and the patient suffers from profound prostration. The vomitus is composed of the last ingested food, later bile is ejected and finally the vomited matter resembles pure water. The purging takes place simultaneously, becomes continuous and later assumes the rice-water character of true cholera. Painful cramps often affect the muscles, particularly in the calves of the legs. Thirst, restlessness, a cold and clammy skin and a rapid pulse follow the seizures. Fever is not high, though the internal heat of the intestines is extreme.

Diagnosis.—The symptoms of this disease being so similar to those of *Asiatic cholera,* it is only necessary to exclude the latter by the absence of an epidemic and the true bacillus. In *irritant poisonings* the gastric symptoms precede the intestinal by several days. Arsenic, antimony and mushroom poisoning must be differentiated.

Prognosis.—The majority of cases respond readily to treatment and many times the attacks disappear in a few hours after the stomach and bowels are relieved of the irritating material. Treatment is, however, an important essential, else the disease may terminate in a chronic gastro-enteritis. The aged and the very young may succumb to a severe attack.

Treatment.—When the stomach and upper intestine are first affected, it is a safe procedure to relieve the bowel by a mild cathartic. A saline may answer for this purpose, though it is often best to employ an agent like cascara, castor oil or calomel. The alimentary canal must then be given as complete a rest as possible. No food is necessary for twenty-four or forty-eight hours. Then the blandest liquids may be prescribed at short intervals until it is safe to resort to a light solid diet. Hot water as a beverage gives much relief and bits of ice may quiet the gastric excitement. Carbonated waters or small doses of champagne may be used in some cases. Intestinal antiseptics, as advised under the head of acute catarrhal enteritis, should be employed. .

When *pain and diarrhea* are severe and persistent, some form of opium is indicated. Severe cases will require an injection of morphin (gr. 1/8-1/4) and this may be repeated at intervals of two or three hours until the peristaltic excitement is completely overcome. Codein, in the first decimal potency, is often tolerated better than morphin, and chlorodyne tablets are valuable agents in this condition. The cholera mixtures, containing chloroform, are serviceable in severe cases, and the compounds containing opium and camphor are sometimes useful. *Local applications* of heat give adjuvant relief and for this there is nothing better than the mustard plaster. High salines, with the water at least 100° F. soothe the intestine and alleviate the pain.

The *internal medication* gives the most satisfactory help. *Camphor* in a low potency, is one of our best agents. It is indicated when cramps in the bowels and limbs are attended by coldness, prostration and a tendency to collapse. *Colocynth, dioscorea, chamomilla, cantharis* and *plumbum* may be compared in accordance with the symptoms previously mentioned. *Cuprum ars.* is most frequently indicated. It covers the symptoms of excessive nausea with vomiting, violent paroxysmal cramps in the stomach and bowels, with a tense and tender abdomen. The stools are green and decidedly watery and the movements are followed by pain and tenesmus. *Arsenicum alb.* is to be thought of when pain is not so pronounced and the gastric disturbance is aggravated. *Antimonium crudum* will be indicated when thirst, nausea and vomiting and alternate constipation and diarrhea suggest a long existing gastro-enteritis. *Ipecac* in minute doses will usually correct the vomiting, and it has some action upon the intestinal unrest. *Veratrum alb.* has many of the symptoms of cuprum and camphor pertaining to the bowels and in addition it covers the thirst for cold drinks with immediate vomiting as soon as cold water is taken into the stomach. *Euphorbium* has symptoms similar to cuprum, and *iris versicolor, chelidonium* and *chionanthus* refer to hepatic complications. *Podophyllum* helps in the recurrent cases when the green, fetid and watery movements are attended by very slight pain. *Aloes, mercurius* and *sulphur* should be compared.

DIARRHEAS IN CHILDREN.

Various terms have been employed to characterize the acute gastro-intestinal disturbances which occur among children, particularly during the summer months. We may best study them under the following heads: Acute dyspeptic enteritis, acute enterocolitis and cholera infantum.

ACUTE DYSPEPTIC ENTERITIS.

This is a disease which arises from dietetic errors in children and is characterized by an acute inflammation of the small intestine. For the most part we observe these conditions when the prepared foods are either impure, indigestible or abnormal. Too frequently the cause is due to a filthy bottle. Many times overfeeding is a factor to be considered and we may often find that the mother's milk is perverted by emotional causes or improper diet. The development of bacteria and their toxins are the most common irritants either before or after the food has entered the stomach. The *bacterium aërogenes* is often found in the upper bowel when there has been an exclusive milk diet and the *bacillus coli communis* thrives, under unfavorable conditions, in the lower part of the small intestine. The *fermentation* caused by these and possibly other bacteria are the underlying factors in this disease. Imperfect dentition, the extreme heat of summer, filth and a predisposing constitutional weakness must be regarded as contributing causes. The morbid anatomy will be defined by a catarrhal swelling in the early stages of enteritis.

Symptoms.—*Restlessness,* thirst, fever, nausea and vomiting are early symptoms. The temperature, though slight in the majority of cases, may rise suddenly to 103° or 104° F. *Vomiting* may take place, though some cases may show no gastric disturbance. *Purging* usually appears as a primary symptom. The stools do not at first show much change from normal. As they become more frequent a watery appearance is observed and milk curds may be in evidence. The color will vary from a greenish hue to that of "chopped spinach or chopped eggs" and the odor is sour and otherwise offensive. It is not uncommon to find the cause of the disturbance in particles of undigested food. Mucus and blood may appear later, though the latter is not frequently found. *Pain,* colicky in character, is invariably present, but it usually disappears after the evacuation. *Flatulence* is excessive, the urine is scanty and evidence of general suffering will be detected by the anxious expression of the face and the puny appearance. *Emaciation* becomes pronounced if the disease is protracted.

Diagnosis.—The rather sudden onset is a distinguishing feature. Enterocolitis has more mucus in the stools and cholera infantum has more serous discharges, violent vomiting, more pain, high fever, cool skin and a greater tendency to collapse.

Prognosis.—The outcome of this disease depends much upon the condition of the weather, the constitutional factors, the general hygiene and the kind of food prescribed. Poorly nourished children furnish the greatest mortality.

Treatment.—*Perfect rest* for the alimentary canal is the first essential of treatment. Water, albumen water or barley water should be the only nourishment for some time. The dietetic error should be remedied by all means. When the diet is resumed milk should be peptonized and diluted with sterile water until the digestive function will tolerate the normal quantity. Lime water and often Vichy water may be used as diluents or drinks. If cow's milk is employed, it should be modified in accordance with established rules for the preparation of such foods. If the mother's milk is continued, careful investigation should be made to find the cause of its indigestibility. The *next object of treatment* is the careful consideration of hygienic conditions and the proper cleansing of the bottle. One person only should prepare the child's food so that there may be perfect familiarity with this duty. It will be best also to exercise great caution lest some interested member of the family assume the privilege of giving the baby a taste of some harmful food. It is of equal importance to see that the child is taken out regularly into the fresh air and sunshine, properly bathed, clothed according to the requirements of the season and regularly put to bed. Few people seem to realize that a child is human like the rest of mankind.

Under *medicinal treatment* it is proper to consider first of all, the probable cause. As the food is most commonly at fault a mild cathartic is not out of place. Some prefer for this the divided doses of calomel (gr. 1/10) with soda until one-half or one grain is administered. Castor oil or olive oil are also of great value for this purpose. Bismuth and prepared chalk, in two grain doses, are often prescribed every two or three hours until the intestinal indigestion is relieved. Some resort to the antiseptic use of salol, but this has not been generally commended. Paregoric and other mild opiates are the panaceas of grandmothers and other interlopers, but modern medicine does not encourage such practice except as a last resort. The minimum amount of medicine should be used in all of these cases.

The internal remedies to be thought of first relate to the fever. *Aconite, belladonna* and *chamomilla* will generally suffice in this respect. *Ipecac, pulsatilla* and *podophyllum* will often correct a more persistent fever which is dependent upon the intestinal intoxication. The remedies for the colicky pain will be similar to those in the enteritis of adults and the diarrhea will usually respond to the medication which overcomes the intestinal fermentation. *Cuprum ars., arsenicum alb., colocynth, dioscorea,* etc., may be employed as previously suggested. *Bismuth subgallate, pepsin* and *papoid,* in one to three grain doses, are serviceable as digestive aids. *Capsicum* often overcomes the pain and tenesmus when mucus and blood are found in the stools. *Colchicum* is a rational remedy for autumnal dysentery, should that be a complication. We should also keep in mind *nitric acid* and *mercurius* when their indications are present. *Borax* is a remedy which corresponds to the symptoms of abdominal pain and distention when an apthous condition of the mouth exists and the stools are pappy, loose and offensive. *Bryonia* is also a valuable remedy in this disease as it pertains to both gastric and intestinal complications. The abdomen is the seat of sharp, stitching pain, while the passages are brown in color and partially constipated. *Veratrum album* should never be forgotten in this condition. *Sulphur* is probably our most useful remedy not alone for the diarrheic conditions, but more particularly because it has a more deepseated action relating to the underlying dyscrasia. *Calcarea carb.*, *calc. phos.* and *calc. iodid* may also be counted upon as helpful constitutional remedies.

ACUTE ENTEROCOLITIS.

The synonyms for this disease are acute ileocolitis, follicular enteritis and follicular dysentery. The inflammatory process is much more severe than in dyspeptic enteritis, the lesion is in the ileum and colon and the lymph follicles are simultaneously affected. The causes are similar to those of dyspeptic enteritis and it is often a sequela of this disturbance or cholera infantum. It is most common during the summer, though it may occur in cool weather. Early babyhood or the first to third year of childhood are the most favorable periods for its occurrence.

Pathology.—This is found chiefly in the ileum and colon. At first the mucous membrane is congested and swollen. Peyer's patches and the solitary follicles are enlarged and the epithelium soon begins to exfoliate in spots. After the first week the solitary follicles and Peyer's patches show signs of ulceration. Later the inflammation affects more of the mucosa, the ulcers enlarge and invade the muscular coats, while sloughing takes place. A diffuse infiltration of the bowel leads to considerable thickening which may alter the normal structure of the part to a considerable extent. Coagulation necrosis may take place.

Symptoms.—The symptoms are similar, in many respects, to those of dyspeptic diarrhea. The *fever* is, however, more pronounced as it often rises rapidly to 104° F. or higher; it is not generally so high as in cholera infantum. Vomiting is not common, but the pain is quite severe and the abdomen becomes swollen and tense. The *fecal discharges* are small in quantity, but frequent; mucus is in abundance and blood may be present. The symptoms show a tendency to abate during the early stages and prompt recovery is possible. Should the fever persist, we may expect a prolonged course of the disease, the passages will be more frequent, painful and bloody. Under this condition the odorless stools of the ordinary case will be changed into those of extremely fetid character. The urine becomes scanty, emaciation is pronounced, and a fatal termination may be expected at any time. In the severe cases, if death does not take place suddenly, we may look for a prolonged convalescence.

Diagnosis.—The severe pain, the high fever, the abundance of mucus and the rapid prostration will distinguish this from dyspeptic diarrhea. Cholera infantum will show a higher fever, obstinate vomiting, serous stools and collapse.

Prognosis.—The average case will respond to treatment more favorably than either dyspeptic diarrhea or cholera infantum. Much, however, depends upon the prompt treatment and a careful limitation of the diet. Protracted fever, bloody stools and persistent abdominal pain must be regarded with apprehension.

Treatment.—The hygienic and dietetic demands outlined under dyspeptic diarrhea must be followed out with greatest care. The general and medicinal treatment should conform quite closely to the suggestions under cholera infantum. Anodynes are probably necessary in the early stages in order to limit the congestive progress. These, however, should be used with greatest caution inasmuch as they may subvert the natural bowel elimination. Flushing of the colon with normal salt solution will be helpful in some cases, though this measure should not be instituted indiscriminately. Low rectal enemas with astringent solutions are often palliative.

CHOLERA INFANTUM.

This is a form of acute catarrhal enteritis of intense severity which resembles the cholera morbus of adults or presents the symptoms which are typical of sporadic cholera. It is due to a marked gastro-intestinal irritation which is caused by the toxins of decomposed and fermented food. It occurs in young children and almost invariably the source of trouble may be traced to infected milk. A specific germ has not yet been discovered. It generally occurs during the summer months and the constitutional conditions which pervert the digestion are predisposing factors.

Pathology.—A definite organism has not been found. The various bacteria which are liable to exist in the intestine become virile through the agency of infected milk and the heat of summer. The mucous membrane shows evidence of a highly irritating poison.

Symptoms.—The *onset* is sudden and the disease is characterized by severe vomiting and purging, together with rapidly developing constitutional symptoms. The *vomited matter* consists at first of the food and later of mucus and bile. The *stools* are copious; in the beginning they are fecal and offensive, later they assume a greenish character and finally they become watery and are ejected with much force. *Pain* is severe, the abdomen is rigid, the limbs are flexed and a general convulsion is recognized. *Fever* is extreme, the temperature often reaching 105° F., the pulse is frequent and feeble, thirst is pronounced and the child is very restless and nervous. The surface of the body is cold and emaciation is rapid. Very soon the state of restlessness yields to apathy and indifference until collapse appears. The patient may pass into a state of torpor or semi-consciousness with frequent convulsive seizures. The head will be retracted, Cheyne-Stokes respiration is liable to appear, the pupils may become irregular and serious brain symptoms are liable to arise, all of which symptoms point to sudden death. In favorable cases the characteristic symptoms subside gradually and recovery may take place.

Diagnosis.—The differentiation from the ordinary summer diarrheas ought to be made without difficulty. The serous vomiting and purging, the high temperature, rapid emaciation, prostration and nervous symptoms are sufficiently characteristic for a diagnosis.

Prognosis.—A grave prognosis may be rendered unless there is a gradual abatement of the severe symptoms in twenty-four to forty-eight hours. Very young infants rarely get well and the extreme hot weather is an unfavorable feature. Protracted high temperature, intense prostration and marked nervous symptoms presage an early death. It is possible for the disease to merge into a typical enterocolitis.

Treatment.—*Preventive treatment* is not a possibility after the disease is once established. Dietetic considerations which pertain to a careful preparation and administration of sterilized milk is, however, necessary to avoid alimentary irritation. Parents and nurses should always anticipate the danger during the summer months particularly. In the first stages the digestive organs should be given absolute rest for at least twenty-four hours. The food should then be prepared in accordance with well established principles of infant feeding. Albumen water, barley water and light broths may be tried as the severe symptoms subside. Warm distilled water, if taken freely, may serve to cleanse the stomach and aid in eliminating the toxins. Enemas of hot water may give a similar relief to the intestine, though these should be cautiously employed.

Opiates are advised by many experienced practicians, though it is always a question whether these should be used for fear of functional suppression and the retention of toxins. Injections of morphin (gr. 1/8-1/2) have been employed successfully, but are rarely needed. Deodorized opium—two to four drops in an ounce of starch water—may be injected into the rectum. Dover's powder in small doses is often very valuable.

The *adjuvant treatment* embraces the cool bath, the pack or the systematic sponging as in other forms of fever. In case of collapse the hot bath should be substituted. Stimulants may be demanded, and for this a little brandy or whiskey may be required. Strychnia (gr. 1/100-1/60) may be given hypodermatically. Such intestinal antiseptics as the sulphocarbolates, salol, beta napthol and bismuth subnitrate should not be overlooked in these cases.

The internal medication will include several well established remedies. *Arsenicum alb.*. is one of the most serviceable, inasmuch as its symptomatology is similar to the symptoms of the disease. The vomiting and purging, the thirst, restlessness and depression all point to its indications. *Cuprum ars.,* may be a better remedy when the abdominal pains are more severe and when the disease simulates the characteristic features of cholera. *Veratrum alb.* is the next important remedy. Its symptomatology is a perfect picture of cholera; the vomiting, purging, weakness, coldness, pain in the abdomen preceding a stool, the cramps in the limbs, the cold perspiration and other symptoms complete the clinical picture. It is my belief also that it should be used in appreciable doses and the dose should be frequently repeated. *Camphor* is the next important remedy, and relates to extreme collapse with an icy coldness of the whole body; chilliness is more characteristic than in the other remedies; cramps also affect the limbs; the diarrhea is attended by colic and the stools are often involuntary. *Terebinth* may be compared, but when this is indicated there will be more abdominal distention with nephritic complications.

Secale has also many similar symptoms, but the stools are often involuntary and hemorrhage may be observed. Should the stools alternate between diarrhea and constipation, with a tendency to involuntary action, we may think of *opium* in potency. *Hyoscyamus* covers many of the nervous symptoms and it refers also to the colic and tympanites which occur with the diarrhea. *Hydrocyanic acid* will be useful when intestinal paresis follows the long-continued choleraic features.

For the febrile symptoms we shall obtain much help from *aconite* during the first stages of restless excitement, from *belladonna* when the griping, tenesmus and greenish stools are associated with a fever, which denotes a general septic condition. *Baptisia* and *rhus tox* may be recognized when the typhoid state ensues. *Chamomilla* is indicated when the colic, flatulence and fretfulness are marked. *Podophyllum* will sometimes be suggested by the thin, copious and painless stools. *Zincum met.* and *zincum cyanid* may be used when the collapse continues after the vomiting and diarrhea cease. *Ferrum phos.* is another remedy which should never be overlooked in any disease of childhood.

Croton tiglium often aborts the disease, inasmuch as it corrects the copious watery stools which are ejected with great force. The danger from its use must always be considered as it may suppress a diarrhea and cause severe systemic reactions. *Capsicum* and *cantharis* are allied with this and often relieve the excessive serous passages and the violent abdominal pains. *Antimonium crudum, ipecac, iris, nux vomica* and *sulphur* may be utilized as symptomatology may direct.

CELIAC DISEASE.

This is also called diarrhea alba and diarrhea chylosa. It is a form of intestinal catarrh occurring in children at the age of one to five years. It is marked by copious, fetid and frothy discharges which resemble oatmeal gruel in color and consistency.

The *pathology* of the disease is not known, though it has been found in association with intestinal ulcers. The filaria sanguinis hominis has been found in the feces. Osler claims it resembles the disease which is known as "hill diarrhea" or the "white flux" of India.

The *symptoms,* in addition to the characteristic diarrhea, are slow in development and represent a progressive weakness and emaciation. There is no fever, but the abdomen is moderately distended and has a doughy and inelastic feeling on palpation. It is a fatal disease and little may be expected from treatment.

PSEUDO-MEMBRANOUS ENTERITIS.

This is an intense inflammation of the intestinal mucosa which is followed by the formation of a false membrane. It has otherwise been defined as croupous enteritis or diphtheritic enteritis. It is not a common disease. We may discover it in connection with infectious diseases like pyemia, pneumonia, scarlet fever and typhoid. Mechanical irritants like impacted feces, enteroliths and gallstones, chemical irritants like ammonia, acids, mercury and arsenic, are often the causes to which it may be traced. Constitutional diseases also have much to do with its development.

Pathology.—The membrane varies in extent and thickness. It has the appearance of a croupous deposit which is grayish or grayish-yellow in color. The lesion is most frequently in the colon, though there are cases in which the solitary follicles are alone inflamed and covered with the deposit.

Symptoms.—When an irritant poison is the cause we may expect vomiting and purging, but in other cases we shall not observe many symptoms aside from the croupous exudate which passes from the rectum.

Treatment.—For the most part the result must be attained by symptomatic treatment and a general correction of nutrition and digestion. Pain may be greatly relieved by hot applications and large enemas. Opiates will be required in severe cases. *Mercurius, iodin, iodid of lime, kali bichromicum, hepar sulphur, hydriodic acid* and similar remedies will be useful.

PHLEGMONOUS ENTERITIS.

This is a rare disease which is similar to the phlegmonous involvement of the stomach. It is a diffuse suppurative inflammation of the submucosa. In exceptional cases it appears as a circumscribed abscess. The exanthemata and aseptic conditions are traceable causes. It has been found also after hernia and intussusception.

Symptoms.—These closely resemble those of peritonitis. Vomiting is often severe and it may become stercoraceous. If there is obstruction, pain and tenesmus will be severe. Rigors often precede the fever, which is unusually high. Prostration and collapse may precede death.

Treatment.—Internal medication will serve only to alleviate the fever and sustain the strength. The surgeon will be needed in case of abscess formation or intestinal obstruction.

SPRUE.

Sprue or psilosis is a disease of which we know but little in this country. We are indebted for our knowledge of it to Manson, who describes it as "an insidious, chronic, remitting inflammation of a whole or a part of the mucous membrane of the alimentary canal. It occurs principally with those who reside in tropical or subtropical climates."

The *pathologic features* are defined as "a patchy or general destruction on the surface of the mucosa which varies from a slight erosion to an extensive disintegration of the villi, glands and follicles." The etiology is not known, though it originates, no doubt, from a micro-organism.

The *symptoms,* as defined by Manson, are "irregular action of the bowels, the passage of copious, pale, drab-colored, yeasty looking, sickly smelling stools. The complexion is dark or muddy, the abdomen is distended and emaciation is marked." No treatment has yet been settled upon other than the correction of dietetic errors. Several cases have been reported recently as markedly relieved by a strawberry diet.

HEMORRHAGIC INFARCT OF THE BOWEL.

Hemorrhagic extravasation into the wall of the small intestine occurs as the result of embolism or thrombosis of the mesenteric arteries. Endocardial vegetations or clots from an aortic aneurysm are also causes. The symptoms are grave and the termination in sudden death generally takes place. Pain, vomiting, constipation and abdominal distention are the characteristic features.

INTESTINAL ULCERATION.

As a feature of other diseases this has been mentioned under the subjects of typhoid fever, dysentery, tuberculosis, enteritis and duodenal ulcers. The typical evidence of its existence is hemorrhage and this is the symptom which first demands attention. Primary tuberculous ulcer may exist for some time in the ileum or appendix and not manifest any marked signs of its presence until hemorrhage or other general symptoms appear. Syphilitic ulcers are found in the rectum and this would suggest a similar invasion of the colon. Embolic ulcer may exist in the intestine when a valvular cardiac lesion or a septic focus furnishes the embolic plug.

There are other factors which lead to the localized ulceration of the intestine, though these are usually included in the pathologic descriptions under different topics. Impacted feces and foreign matter may be the exciting causes many times. The mucous membrane of the intestine is easily torn and localized irritation of this kind may establish the ulcerative process. Diseases of the intestinal walls, diseases of the blood or any visceral diseases which increase the blood pressure within the bowel are causes of hemorrhage and a possible subsequent ulceration. Amyloid degenerations, aneurysm and all intestinal malignant diseases may be recognized, at some stage, by the rectal discharge of blood and pus; if these symptoms become chronic, they will indicate a condition of ulceration.

Symptoms.—Outside of the one diagnostic sign of hemorrhage, and the possible discharge of pus, there is little in the symptomatology to indicate the presence of ulceration. The pallor, the weak pulse and faintness, and the signs of the predisposing disease may, together with localized abdominal pain, suggest the hemorrhage before blood appears in the stools. The blood which comes from the upper intestine will have a tarry appearance when it passes from the rectum, while the blood coming from the large intestine resembles a fresh coagulum. It is always necessary to exclude hemorrhoidal bleeding or the possible discoloration coming from a food product.

Prognosis.—The course of the disease depends upon the location and extent of the ulceration. This naturally comprehends the pathology of the causative malady. Persistent or copious hemorrhage is always dangerous.

Treatment.—In order to cure the disease we must remove the cause and this relates to the complicating factors. This phase of study and treatment is taken up under such subjects as typhoid and dysentery. The ulcerations and hemorrhages which take place in the upper bowel are reached only by the internal remedy or prompt surgical intervention. We may help the chronic ulcerations of the lower bowel by medicated enemas, if the hemorrhage is not active, or by means of a sigmoid speculum we may apply our medicament directly to the ulcerated spot or near enough to affect it by absorption.

The hemorrhage may be stopped by remedies administered for that purpose. It will be necessary, in addition, to resort to all the expedients mentioned under the treatment of typhoid fever. *Hamamelis* is probably our best remedy for active hemorrhage; the supposition has always been that it is more useful for passive hemorrhage, but I have used it for all forms of bleeding and have found it efficacious. In severe cases it should be used in teaspoonful doses of the aqueous solution, but in the average cases five to ten drops of the tincture is sufficient though, at first, it should be repeated often. The remedies to be compared are *arnica, calendula, trillium, geranium* and *hydrangea. Erigeron* has been used for both active and for slight bowel hemorrhages; it is serviceable also in slight hemorrhoidal oozing and may be continued for some time in potency when the stools are streaked with blood. *Ipecac* is a standard remedy for profuse and bright red hemorrhage, but it is usually associated with some gastric perversion, as nausea is a prevailing characteristic. *Terebinth* is our best remedy when ulceration is extensive. We have found this true in typhoid where it is employed as the emulsion in the enema and internally in the third decimal potency. *Nitric acid, alumen* and *secale* may be compared. *Millefolium* has been successfully used for profuse hemorrhage of bright red color attended with vertigo and palpitation which may attend chronic enteritis. *Arsenicum, cinchona* and *phosphorus* have each their special sphere of action, but some form of anemia is the underlying complication. *Ferrum* points to a blood change and *sulphur* refers to a dyscrasia; yet each within its limitations is of service in chronic stages. *Crotalus,* like *leptandrin,* is indicated by the black stools which have the coffee-ground appearance. *Naja* and *lachesis* are similar in their general reference to cardiac insufficiency. The scanty stool with bloody mucus and bile is always indicative of imperfect biliary action and, therefore, *mercurius dulcis,* in quarter grain doses an hour apart, until a grain is taken, may not be amiss. In protracted cases it may be advisable to use it in the third decimal potency. One of the best remedies of recent experience is *hydrastin hydrochlorate.* This will give good results in both active and passive hemorrhage, and it should be used in the second decimal potency every two hours until there is an absolute cessation of all signs of bleeding. It may be employed hypodermatically in doses of one-eighth grain. Stypticin (1-4 gr. t. d.) may be used for its hemostatic effect.

INTESTINAL OBSTRUCTION.

Occlusion of the intestinal canal sufficient to cause an obstruction to the passage of feces characterizes this condition. It may refer, however, to pathologic conditions which have a more serious train of symptoms and, in some of these cases the free passage of feces is not always interrupted. It is perhaps correct to include under this same head the deformities, like imperforate rectum, which are purely congenital. The classification may also be made to cover both the obstructions due to pressure upon the intestine from without and those which result in constriction from a pathologic lesion of the intestinal wall. The occlusion may be partial or complete and the cases may also be classed as acute or chronic. A more complete study under distinctive titles may be undertaken as follows:

INTERNAL STRANGULATION.

Hernia within the abdomen or internal strangulation of the bowel is a form of acute intestinal obstruction which is caused by inflammatory bands or adhesions of adjacent tissues. These are commonly produced by previous attacks of peritonitis, abdominal injury and rarely by pelvic operations. It is without doubt one of the most frequent forms of acute obstruction. Males are affected more than females and a fair proportion of cases occur during the early period of life; when it is an incident of youth it is usually caused by vitelline remains. The strangulation is located, in a majority of instances, in the small intestine and the right iliac fossa is the most common seat of occurrence.

The constriction is made by fibrous bands forming a loop or a knot which incloses a portion of the ileum. The free end of Meckel's diverticulum may become attached to the abdominal wall and cause a stricture of an intestinal loop. The remains of vitelline blood vessels may likewise constrict the bowel and the same result may follow in case of an adhesive attachment of the free end of the appendix. Strangulation may also follow when the intestine is pushed through an omental or mesenteric slit or crowded back into a peritoneal pouch. An adherent Fallopian tube or pedunculated tumors may cause strangulation by their pressure upon the intestine. Diaphragmatic hernia may exist when there is a congenital or traumatic imperfection in the anatomy of this part. When the strangulated intestine is deprived of nourishment gangrene ensues.

INTUSSUSCEPTION.

Invagination of the intestine is a condition of obstruction due to the telescoping of one section of the bowel into another, generally from above downward. It is caused by circumscribed or irregular peristalsis. Nothnagel describes it as a "thrusting forward of the receiving portion by a contraction of the longitudinal muscular coat or a thrusting inward and downward of the portion immediately above by means of an increased or spasmodic peristaltic action, thus forming a cylindrical or sausage-shaped tumor varying from half an inch to a foot or more in length." The external or receiving portion of the intestine is called the *intussuscipiens,* the mucous surface of which is in contact with the mucous surface of the *intussusceptum* or invaginated portion of the bowel. The peritoneal surface of the latter is in contact with the peritoneal surface of the internal or returning portion. The two mucous surfaces of the returning portion are also in apposition.

Intussusception may take place in any part of the bowel, though the most frequent location is in the ileocecal area. The terms employed are *ileocecal* when the ileocecal valve passes into the colon; *ileocolic* when the ileum passes into the cecum or colon through the ileocecal valve; *colic* when one portion of the colon enters a succeeding portion; *colicorectal* when a part of the colon enters the rectum. The *ileocecal* and the *ileocolic* are most common.

We may regard this form of intestinal obstruction as next to strangulation in point of frequency. It is observed mostly in the male sex and it is quite common among children under ten years of age. Diarrhea and habitual constipation are predisposing causes. There is also a form of intussusception which often takes place a short time before death, due, no doubt, to irregular and excited peristalsis during the death struggle. The autopsy alone reveals this.

During the early stages of invagination the intestine may, as a rule, be returned to its normal condition, but if that is not done in the beginning, it becomes a difficult procedure on account of the tumefaction and peritoneal adhesions. The secondary inflammatory changes lead to functional and nutritional disturbances which make it a serious disease.

VOLVULUS.

Twists or knots in the intestine constitute occasional forms of obstruction and they appear most frequently in elderly people. Obstinate constipation may be a predisposing complication. The bowel is twisted on its mesenteric axis and the lesions usually are located in the sigmoid flexure, while a small proportion occur in the ileocecal and cecal region. The occluded part of the bowel becomes distended with feces and gas while the resulting congestion and inflammation tend to maintain the abnormal condition. This condition is increased by further peritoneal inflammation. Knots may be formed by loops of the ileum or it may become twisted about the cecum as a pedicle.

STRICTURES AND MORBID GROWTHS.

Both of these forms of chronic obstruction occur in advanced life and women are afflicted more often than men. The large intestine is the most common seat of the lesion, the majority being found in the left iliac fossa.

Stricture is either congenital or acquired. The former is defined by an imperforate anus or a narrowing of the sigmoid. It may also occur as an imperfect union of the pylorus and duodenum. Acquired stricture is usually the result of cicatricial formations following the healing of an ulcer, or it may result from cancerous infiltration of the intestinal wall. Typhoid fever, dysentery, intestinal ulceration associated with enteritis, tuberculosis or syphilis, are exciting factors. Traumatism and strangulated hernia are occasional causes.

Morbid growths outside of the bowel may create an obstruction by pressure. Cancer may invade the intestinal structure itself and thus decrease its caliber. The rectum and sigmoid are the regions generally invaded. Cylindric-celled epithelioma is liable to form an annular occlusion of the rectum or lower part of the sigmoid. Colloid cancer is sometimes the pathologic cause.

Intestinal paralysis should also be included under this head inasmuch as it is a chronic form of obstruction though functional in character. It is either circumscribed or diffuse and follows from such inflammatory disturbances as peritonitis or enteritis. It is not uncommonly a result of excessive handling during abdominal operations.

OBSTRUCTION FROM ABNORMAL CONTENTS OR FOREIGN BODIES.

Gallstones may enter the bowel by ulcerating through the gallbladder, or by passing through the common duct; they may lodge in the ileocecal region or, less frequently, in the small intestine. Enteroliths composed of phosphate of lime and magnesia, with a foreign body as a nucleus, are occasional causes of obstruction; they lodge in diverticula or in the appendix.

Foreign bodies like buttons, pins, coins, fruit stones and even false teeth are sometimes swallowed and cause intestinal obstruction.

FECAL OBSTRUCTION.

This is a common form of intestinal occlusion and we find its occurrence most frequently among women or in those who live a sedentary or inactive life. The large intestine is generally implicated and the lower part is the seat of impaction oftener than the upper. The cecum offers the greatest opportunity for paralytic involvement as it is a point where pressure is concentrated from above and below. The ileum may also be involved and the distended or paralytic condition of the intestine may be favored by an associate development of local peritonitis. Hypertrophy of the intestinal wall above the point of obstruction is always enhanced by the increased peristalsis induced in the effort to overcome the impaction.

Constipation is the factor which always favors this form of obstruction. *Congenital abnormalities* may be regarded as a cause, and habit has much to do with the development of pathologic conditions which favor it. *Enteritis* and *peritonitis* may weaken the muscular power of the intestine and the resultant imperfection in the peristaltic wave may lead to the paralysis of a segment which suddenly arrests the free passage of the feces. The fecal plug becomes harder as it is retained until the stoppage is complete and the bowel function is entirely arrested.

Pathology.—The changes which are found in nearly every case of intestinal obstruction are similar. First there is a dilatation above the point of obstruction and this will be attended by some tissue hypertrophy if the case becomes chronic. The intestine below this point is empty and there may follow an atrophy or narrowing of the caliber as the result of inaction. The intestinal wall, in the region of the obstruction, becomes inflamed and there will be a localized peritonitis which is either acute or chronic in character. Catarrhal inflammation of the mucous membrane is a natural result and an ulcerative or gangrenous process may ensue, while the danger of a general peritonitis or perforation is always present.

General Symptoms.—*Acute obstruction.* Severe abdominal *pain* is the first complaint. It appears suddenly and is constant, though it may have periods of exacerbation. It is most frequently located near the umbilicus, but it seems to have no relevancy to the location of the obstruction. It is generally preceded by *diarrhea* or *constipation* and there is often an unpleasant abdominal sensation some time before the severe paroxysm. There are exceptional cases in which pain is not a marked feature.

Vomiting appears nearly as early as the pain. Nausea and painful retching are often pronounced. The vomitus at first consists of food and mucus; later it has a bilious character until finally it becomes stercoraceous. Volvulus, stricture and tumor are more liable to cause vomiting than strangulation and intussusception. The *severity of the paroxysm* generally increases as the obstruction continues and it invariably lasts until there is relief by a normal passage of feces, or death ensues. Hiccough is an occasional symptom, particularly in volvulus. *Tenderness* is apparent when localized peritonitis is established, but does not exist until a few days after the initial lesion. *Tympany* is next observed and is more marked when the large intestine is the seat of trouble.

Flatus is incarcerated and constipation is absolute, though at first there may be discharges of feces until the intestine is emptied below the point of obstruction. A *tumor* or swelling characterized by absolute dulness is an occasional physical sign and an intestinal coil may present a visible protuberance. The tumor of intussusception will often be detected in the left iliac region and morbid growths may sometimes be felt through the rectum. *Exaggerated peristalsis* above the point of obstruction is often recognized by inspection, and borborygmi, gurgling and splashing sounds may be heard on auscultation.

The *constitutional* symptoms are conspicuous from the beginning. Prostration is pronounced and is liable to terminate in sudden collapse. The patient is restless, the features are pinched, the face is cold, the eyes are sunken and the body is bathed in cold perspiration. The pulse is weak and rapid and the temperature is subnormal until peritonitis sets in, when it is elevated. Respiration is rapid and the urine is diminished or may be suppressed.

Chronic Obstruction. The symptoms of the chronic variety resemble those of the acute form, but are not so distinctive. There may be simply the history of chronic constipation with occasional alternation of diarrheic attacks. A complete retention of feces may finally take place. If this persists the abdomen swells gradually and pain becomes localized. The general health is impaired, the appetite fails, the tongue becomes coated and the breath offensive. In some instances lethargy may follow the progressive weakness. The impaction may give rise to a fecal tumor which may be felt as a hard or a soft and yielding mass frequently in the right iliac region. If it is farther up, it may be mistaken for an enlarged liver or gall bladder, a tumor of the stomach, the spleen or kidneys. In the descending colon it is harder and resembles scybala. Should the abdominal pain become paroxysmal and vomiting ensue, the characteristic features of the acute form may suddenly terminate in death, unless operative relief is obtained.

Diagnosis.—All experienced physicians will agree that the importance of early diagnosis is of first importance from the fact that life is only saved by prompt surgical attention. The decision must be made as to the actual existence of an obstruction, its location and its cause. The symptoms of sudden and severe pain in the abdomen, fecal vomiting, complete constipation, tympanites and marked depression will generally intimate that the cause is acute intestinal obstruction.

The *location of the obstruction* is a difficult matter to decide unless there is positive evidence of a tumor. *Active peristalsis* above a definite point would indicate the seat of the lesion to a certain extent. *Pain* affords little satisfaction in diagnosis, as it is rarely at the seat of obstruction. When the *small intestine is obstructed* vomiting begins early, is at first scanty and then becomes fecal. When the *large intestine is involved* there is less vomiting and it is not so liable to be fecal. The tympanites is marked, is more central and associated with tenesmus and bloody stools when the large intestine is implicated. When the lesion is located in the small intestine distention is not so pronounced and is higher in the abdomen. Much help in making a decision is obtained by locating a tumor or a stricture by means of rectal and vaginal examinations. Also when the rectum remains free of feces we may conclude that the obstruction is in the small intestine. The use of the enema may give some information as to the height of the lesion by the amount of water which may be injected. When the stenosis is in the duodenum or jejunum it is possible to find an increased amount of indican in the urine, which suggests a stagnation in the upper intestine and the consequent decomposition of albuminous substances. This test will be negative when the large intestine is involved.

The nature of the obstruction will demand a careful study of the history and associate symptoms. *Strangulation* will furnish a previous history of peritonitis, the symptoms are sudden, fecal vomiting appears early and constipation is absolute. Young adults are usually affected. *Intussusception* occurs most frequently with children; it is announced by sudden colicky pain, tenesmus and the presence of mucus and blood in the stools. An elongated cylindric tumor is found in the right iliac or umbilical regions and a digital examination may, in some instances, detect it per rectum. Absolute constipation is unusual. *Volvulus* gives the history of former constipation and it affects those of advanced years. Intestinal atony, pronounced tympany and tenderness appear early. Vomiting is a late symptom and is rarely fecal. The abdomen is rigid and dyspnea may be experienced as a result of gaseous distention. *Fecal impaction* is of slow onset and gives a previous history of habitual constipation. Pain is not so severe, fecal vomiting is uncommon and tympany develops slowly. Palpation over the colon and the digital touch, through the rectum, may reveal the fecal mass. *Obstruction by foreign bodies and calculi* will be detected only through the aid of the history and the symptoms of irritation which point to something of this kind. Physical examination may be of some aid in this respect.

Differential diagnosis must be made first in regard to *enteritis,* which, however, does not develop suddenly, has no fecal vomiting and its fever is characteristic. *Appendicitis* is thoroughly differentiated under that subject. *Acute poisoning* has no fecal vomiting and presents more signs of toxemia. *Biliary colic* is associated with hepatic symptoms, jaundice and some evidence of gallstones or cholesterin in the stools. *Intestinal colic, renal colic* and *displaced kidney* will all furnish diagnostic signs which should be conclusive. *Hernia,* if it becomes strangulated, may be somewhat confusing, but the location, aided by the physical examination, should not make the differentiation difficult.

Acute generalized peritonitis demands careful differentiation. In this we find a history of ulcer, appendicitis or some pelvic infection, while in obstruction there is only a history of previous obstruction or hernia. In peritonitis there is high temperature, but in obstruction no temperature is apparent, except when peritoneal inflammation is established. In peritonitis the pain is continuous and diffuse; vomiting, if it exists, is not stercoraceous, collapse is a symptom in the later stages and there is only a slight increase of indican in the urine. Obstruction presents more localized and paroxysmal pain, vomiting is stercoraceous, collapse is an early sign and indican is excessive in the urine, particularly when the small intestine is affected. In the physical signs peritonitis gives general abdominal distention and no visible peristaltic waves. Obstruction, on the contrary, has little abdominal distention, unless the lesion is in the lower bowel and the peristaltic wave is exaggerated, particularly when the lower intestine is the seat of obstruction. In peritonitis the tenderness is general, effusion is present and the prognosis is invariably unfavorable. In obstruction the tenderness is more localized, effusion is less common and operation gives a favorable prognosis.

Prognosis.—An unfavorable termination may be expected in a few days in acute obstruction, unless operative interference is promptly undertaken. In the chronic form we may expect favorable results when a thorough evacuation is obtained. Secondary peritonitis, gangrene, perforation, pyemia and chronic enteritis make the prognosis unfavorable.

Treatment.—The greatest hope for help rests in early surgical procedures. Medical help is not to be relied upon though atropin in repeated doses hypodermatically has its advocates; nothing is more dangerous than the use of cathartics. Manipulation is to be discouraged, inasmuch as it encourages inflammation and reduction is accomplished only in the early stages. Pain and vomiting may be temporarily relieved by hypodermics of morphin, but this may react unfavorably. Gastric lavage and the withdrawal of all food by mouth may stop the vomiting. Enemas will sometimes relieve the obstruction when it exists in the lower bowel. In the chronic form we may obtain some relief by the treatment of the predisposing cause, but at all times it is best to be prepared for a surgical operation. Massage and electricity are of some adjuvant help after the obstruction has been relieved, but these measures are contraindicated before this is accomplished. Medical treatment other than the use of atropin is absolutely of no value.

CONSTIPATION.

This may be defined as an irregular or insufficient action of the bowels. While it is not in itself a disease, it may arise as a direct or remote result of some disease and if it becomes a chronic condition it may eventually favor the development of a disease. Its serious consequences are often disregarded by both physician and patient. On the other hand there is a tendency, on the part of the laity, to tamper with cathartics when the bowels, if let alone, might naturally correct any lost regularity of evacuation. This indiscriminate disturbance of peristaltic function by the frequent or even occasional use of laxatives is not only unwarranted, but it tends to establish a fixed constipation. In many respects physicians are to be blamed for this inasmuch as a cathartic always makes the patient feel better and it is an easy prescription to make. There is, however, a class of physicians who are unreasonably opposed to such methods when they are necessary. Such practicians, having too much faith in the indicated remedy, are negligent in this duty. When constipation does not yield to regulated diet and regular habits it should be overcome by a mild cathartic, but this should not be continued indefinitely any more than we should persist in cardiac stimulation when the heart is able to take care of itself.

Etiology.—The most common predisposing cause is *irregularity* in attention to this function. This habit occurs most frequently with women and with men in sedentary occupations. Modesty also causes many to refrain from this duty when conveniences are not present. This allows the unnatural accumulation of feces to become hardened into scybala which the propulsive power can not dislodge. More than this, a delayed evacuation of the lower bowel encourages an inactivity and contraction of the sigmoid which overcomes the natural response to the expulsive effort. If such a condition continues for any length of time, a general peristaltic weakness follows. Atony of the bowel may exist as a result of diseases which either affect the bowel or cause a general bodily weakness. *Neurasthenia, hysteria,* spinal diseases, acute fevers and hepatic disorders tend to weaken the bowel function and pervert the normal secretions. Many are subject to torpid livers by inheritance or habit. Too many, however, suffer with a perverted imagination in this respect with the result that they too frequently use cathartics. We must admit that the bile is a necessary stimulant for a natural evacuation, but purgatives do not properly correct the imperfect action of the liver.

Improper food has much to do with constipation. The concentrated foods do not furnish enough residual matter to stimulate peristalsis and too coarse a diet may cause fecal impaction because the residue is in excess. A regular mixed diet containing a sufficient quantity of green vegetables and the coarser cereals is a mandatory requirement. *Another error* is due to the fact that pure water is not used in sufficient quantity to keep the fecal matter semisolid. Again it is possible that the water used may contain those elements which cause constipation. Age is also a factor which often favors constipation. The *aged* always suffer from a peristaltic weakness because degenerative changes occur at that time of life. Infants are often constipated because the milk is too rich in casein, and too little water is given.

Local causes are sometimes at fault. Atony of the abdominal muscles may be caused by obesity or frequent pregnancies. Atony of the large intestine may follow from prolonged colitis. Pressure by tumors and displaced organs, like the uterus, are occasional impediments and strictures are always to be feared in this respect. Hemorrhoids and tight sphincters are local complications which may produce mechanical obstructions in the rectum and hypertrophied rectal valves must not be overlooked.

Symptoms.—The symptoms depend upon the extent of the constipation. Ordinarily headache, vertigo, a bad taste in the mouth, a coated tongue and abdominal or rectal distress are complained of. In the majority of instances the patient suffers only slight inconvenience for several days. Many would not think there was any trouble except from the knowledge that the bowels do not move regularly. I saw one patient in consultation whose bowels had not moved for nearly three weeks. It was supposed to be a case of uremic poisoning as the woman was unconscious. In making a vaginal examination I discovered the distended rectum. When an evacuation was finally obtained, by means of enemas and strong cathartics, the amount passed was something astonishing. *Abdominal pains* may be observed when the retained feces becomes hardened. *Flatus* will often be bothersome and when the stools are voided the rectum will be greatly irritated. The excretion is hard, resembling balls, and is often covered with mucus.

There are *many general symptoms* which result from fecal absorption; these may be enumerated as a sense of malaise, hebetude, cold extremities, anorexia, palpitation and dizziness. Mental depression is the most pronounced phenomenon which attends this functional perversion. *Hemorrhoids,* ulcerative colitis and enteritis are liable to follow a protracted constipation.

Treatment.—Catharsis is the most convenient method of treatment, but it should at all times be avoided if possible. A physician is entirely at fault if he does not resort to every other expedient first. The regular time for an evacuation should be cultivated and kept. The morning is the most natural time, and undue straining should be avoided. The first irregularity should receive careful attention and the cause should be found and removed rather than to produce a movement by the most convenient cathartic. The evacuation should never be sought when in a hurry or under high mental excitement.

The *diet* must receive first consideration. As mentioned under the etiology this, on the average, should comprise a mixed food with a sufficient amount of green vegetables and fruits. Foods which are too stimulating may exhaust the excitability of the intestinal walls. A sufficient amount of food must be introduced into the intestine to cause peristaltic stimulation. This fact is too often overlooked by those who constantly preach about dietetic limitations. The next essential is to take *plenty of pure water* with the meals; this does not mean, however, any encouragement to bolt the food. If the patient does not drink enough water during the day it should be prescribed at stated intervals. The free supply of water to the blood increases the intestinal secretions. Many cases of constipation are due to the loss of fluids through the skin, kidneys or lungs. Diabetes, and certain nervous states, cause this and unless liquid waste is compensated by an adequate increase in the water ingested, the intestinal inactivity will not be overcome. With this in view it may be necessary to discontinue Turkish baths in many instances. When patients are not inclined to drink an adequate amount of water, it is a good principle of practice to prescribe some well known water. The lithia waters and many others which are on the market will answer this purpose, though it is a good idea to occasionally make a change for the mental effect, if for nothing more.

The *enforcement of healthy habits* and the requisite amount of physical exercise is of great importance. The present tendency to over stimulate in every way is bound to have a depressing effect upon the intestinal function and, no doubt, actively predisposes to constipation. Irregular hours for work, as well as for eating, will disturb the normal bowel function and this must not be overlooked in the treatment. The *normal amount of sleep* at the right time is an important prescription for constipation and the physician should not hesitate to prescribe it. Healthful, regular, but not too vigorous *physical exercise* should be taken systematically. It is best to have as much of this in the line of play as possible, but rest must have equal consideration. *Massage,* electricity and osteopathy may supplant this to a considerable extent. The recent interest which has been aroused in the employment of physical culture has cured many patients for us. The habit of *regular bathing* and particularly the cold sponge or the shower every morning has a good stimulating effect.

The *mineral waters* which have aperient action are numerous. Hunyadi, Magnesia, Rubinat, Apenta, Carlsbad and Pluto waters are serviceable in many cases. The objection to their use is due to the fact that they cause too free movements and rectal irritation. When they are properly employed the best action will be obtained by a minimum dose every morning until they may be discontinued. Effervescent *sodium phosphate* is often a sufficient aperient if used regularly. In my practice I get better results by the use of flax seed tea. It may be prepared in this way: Two teaspoonfuls of the unground seed are put into a cup of boiling water every morning; this is covered and allowed to stand until bed-time, when seeds and all are to be taken. I have rarely observed any unfavorable irritation from this and if the method is followed for several weeks, the bowel movements will usually be restored. When the *constipation is more intractable,* bran may be substituted. One to three teaspoonfuls of this may be given in a large cup of hot water at bedtime; bran cakes have also been made for the same purpose. This measure has given me many satisfactory results in extremely chronic cases.

Olive oil and castor oil are standard aperients, though it is not always possible to use them without disturbing the stomach. Castor oil, in one to three dram doses, may be taken in coffee, milk or a little lemon juice to mask the unpleasant taste. Olive oil may be used in larger doses two or three times daily if the stomach will endure it. Either of these may be taken in the juice of a whole orange, several times daily, with good results. In the use of all of these expedients we should gradually reduce the dose until they may be discontinued entirely. The greatest danger is the establishment of the cathartic habit.

Large enemas of water have been advised by many good practicians. As a temporary aid to fecal evacuation there is no doubt as to the efficiency of this measure. That it is a bad habit can not be denied. While they cleanse the colon much better than a cathartic, still their tendency is to weaken the lower peristaltic action and dry the mucous membrane secondarily. Patients who have relied upon this means of help are invariably obliged to continue the practice. In such patients we shall often find the rectum and sigmoid utterly paretic. This is one of the unfortunate after results of a stay in a hospital during the recovery from an operation. Our surgeons and nurses are too prone to the use of this expedient, together with calomel, and when they return the patient to the physician, we are often obliged to work a long time to overcome this habit. Sulphate of magnesia, two ounces, glycerin, two ounces, and turpentine, one half ounce, is a common prescription for a high enema. It may be given when immediate results are demanded. Soap water is used by many good practicians and the results are prompt, but it does not seem wise to use expedients of this kind. Olive oil may be used in large quantities—four to eight ounces—through the rectal tube. This will be efficacious when there is fecal retention of long standing. The use of cold water enemas has been tried for temporary stimulation sufficient to create a prompt movement of the bowels. Perineal and anal douches have also been employed for the same purpose, but the results are not satisfactory or permanent.

In the *mechanical treatment* good results are often obtained by the systematic dilatation of the tight rectal sphincters. This is best performed by the use of the graduated rectal dilators. A better method is to dilate the sigmoid by means of graduated bougies. A method of mine which has given unusually good results is to insert medication above the sigmoid. The procedure is as follows: With an Ives rectal speculum, medication in the form of a twenty to thirty per cent cerate of pine tar, mixed in petroleum, is introduced through the speculum by means of pledgets of absorbent cotton. This is pushed above the rectum with a curved director, and then crowded gently through the sigmoid with a semiflexible bougie. If proper caution is used we may easily insert the cerate into the descending colon. In obstinate cases the treatment should be employed every day for a while and then less frequently until normal movements take place. In this way I have cured many cases which would not respond to other measures. The same treatment is applicable to many conditions of chronic colitis. Suppositories in my experience have accomplished nothing more than temporary relief. Wheat gluten may be of service in the constipation of children. The indiscriminate use of glycerin suppositories should not be considered.

So far as *internal medication* is concerned we are able to prescribe many simple cathartics and laxatives. These, however, invariably contain drugs which are too exciting for permanent results. Anyone can administer a cathartic, but the principle is wrong. I am not yet converted to the use of calomel under all circumstances, unless a powerful cholagogue is needed for emergency practice. If it should be administered, it is best to give it in divided doses of one-tenth to one-quarter grain an hour apart, until a grain or two are given. It will accomplish better and safer results if it is followed by some form of aperient a few hours later. The milder pills containing cascarin, aloin, strychnia and belladonna in small doses are safer expedients and cascara sagrada is always a good alternative when such measures are absolutely needed. Senna tea and milk of magnesia, two drams, two to four times daily, will often correct the most stubborn constipation in children.

I have been an advocate of *chinoidin* as a remedy suitable for most cases of constipation. I use it in two grain pills in the following manner. Two pills are given before each meal and at bed time. The dose is decreased gradually as the movements become normal, then it is to be used intermittently until there is no need of such help. In many cases only two or three pills are required daily. There is no griping or diarrhea from this and the after results are not disturbing. They should, however, be made with a chocolate coating as the gelatin covered pills are liable to become too hard for perfect absorption. It is often advisable to use a pill containing two grains of oxgall, two of pancreatin and one-eighth grain of nux vomica in conjunction with this. In my practice I rarely find it necessary to use any cathartic more powerful than this.

My *favorite plan* of treatment for the cure of chronic constipation is the local treatment previously defined and the use of the flaxseed at bed-time. When this does not accomplish the result I resort to the chinoidin, gradually decreasing the dose until the bowels will respond to the treatment with the aid of flaxseed alone. The local treatment is then performed at longer intervals until the movement is obtained by means of the flaxseed only; when the latter can be omitted the constipation is invariably cured.

Remedies, aside from cathartics, should receive careful attention. If it is possible to find a remedy which represents a symptomatology relating to the definite cause, we should seek to employ it for the sake of cure. Anything which serves as an adjuvant will not be inimical to the required remedy. *Iris versicolor* appeals to me as of first importance in the list. So many cases are due to primary hepatic disorder that its solvent effect is invaluable. I give it, in ten to thirty drops of the tincture, three or four times daily, until the stools become loose; then the dose is gradually lessened. *Chelidonium, carduus, china* and *leptandrin* may be compared. In my experience the last two remedies may be combined with satisfactory results. *Nux vomica* and *strychnia* are naturally considered inasmuch as they have such a decided action upon the involuntary fibres of the intestine and a control over the glandular activity as well. The tonic dose has been employed for many years. The aim of late has been to rely upon the potency. Whether results are possible from this method I can not say. I have sometimes thought I cured my cases in this way. *Sulphur* is of best service when there is morning diarrhea and the evacuation is painless, except for the complication of hemorrhoids, which often exists.

Hydrastis may be prescribed when the stools are covered with mucus, which is an evidence of a low grade inflammation of the intestinal mucous membrane. Rectal prolapse is a common condition when this remedy is indicated and general emaciation and prostration will be detected. The low potencies yield best results and a ten per cent solution of the muriate of hydrastin may be given, in five to ten drop doses, every two or three hours, until a normal action is established. *Plumbum* is indicated when the stools are hard and lumpy and there is much rectal urging. *Alumina* has many similar features and the rectum is very inactive. Either of these may be used in the constipation of elderly people. *Bryonia* also refers to the dryness of mucous membranes and pain is a characteristic feature. The thirst, the feeling of a stone in the stomach, the large and dry stools will confirm the symptomatology. *Opium,* in potency, may be compared to these three remedies inasmuch as it relates to a constipation characterized by hard black balls with a spasmodic retention of the feces alternating often with involuntary actions. *Collinsonia, esculus* and *aloes* have reference to a form of constipation in which the portal circulation is affected. Thus the rectal symptoms are pronounced. The first named will be useful when the disorder comes as the result of pelvic disturbances while the other two pertain to a chronic torpor of the liver or a chronic entero-colitis. *Podophyllum* will often correct a constipation which has been preceded by a diarrhea and this applies particularly in children. *Chionanthus* has a similar action. *Lycopodium* is claimed to be a good remedy when there is an ineffectual urging, and a peristaltic weakness has debilitated the entire alimentary canal. For my part I have had little faith in this remedy, though the experience of others is worthy of consideration.

CARCINOMA OF THE INTESTINE.

A majority of the new growths which invade the intestine are found in the rectum; the cecum and colon are occasionally affected and the small intestine is only occasionally the seat of this morbid process. Compared to gastric carcinoma it is a rare disease. It prevails during the later period of life and antecedent ulcerations may have something to do with its development. It results in intestinal obstruction either by compression or invasion of the intestinal lumen.

Pathology.—Cylindric-celled epithelioma is the most common form of cancer when the growth is located in the sigmoid or the cecum. In the rectum we may find the scirrhus, medullary and colloid cancers. The tumor may be annular in form or it may become a diffuse nodular infiltration. Ulceration is liable to produce a degenerative process and metastatic growths may appear at any time. Benign tumors of the intestine may occur in the form of polypi and fibromata. In exceptional cases we may find lipomas, myomas, angiomas and lymphomas.

Symptoms.—*Distinctive symptoms* are rarely experienced. In some instances the disease is not suggested by the patient's complaints or the physical examination. The *intestinal obstruction* will be noticed first by the gradually increasing constipation and later by the positive signs of obstruction. Very soon the *evidence of a tumor* will develop. *Localized pain* is sometimes a guiding symptom and the intestinal irritation may cause a diarrhea, which alternates with constipation. *Pus, blood* and epithelial debris with gangrenous odor will be found in the stools as the disease progresses. The stools may be ribbon shaped which is always indicative of stenosis. *Anorexia, nausea* and *vomiting* are sometimes attending symptoms.

When the *constitutional effects* are established the peculiar cachexia and the marked emaciation will indicate the probability of a cancerous growth. The *physical signs* are of great value. *Inspection* may sometimes show the presence of a tumor and the peristaltic wave may be observed through the abdominal wall above the carcinomatous obstruction. *Palpation reveals* the nodular conditions and the localized tumor. Boas emphasizes the importance of the sudden appearance and disappearance of small coils of the intestine. *Percussion* will yield dulness or a muffled tympany over the tumor, together with an increasing dulness above, due to the increasing accumulation of feces.

Diagnosis.—In making a diagnosis stress must be laid upon the age, the constitutional symptoms, the localized pain and obstruction, the irregularity of the bowels and the presence of a tumor. Several differentiations must be carefully made. *Cancer of the pylorus* must be differentiated from cancer of the duodenum. The symptoms and the physical findings are decidedly confusing. Both are slightly movable tumors, but in pyloric cancer the gastric obstruction, dilatation and vomiting are more pronounced and appear early in the development of the disease. Duodenal tumors give rise to jaundice, vomiting of bile takes place if the lesion is below the bile papilla, and a fatal hemorrhage may occur. When the stomach is involved we are to place much reliance on the test-meal examination. *Cancer of the head of the pancreas* may also give rise to jaundice, but the tumor is immovable and much deeper seated. When the lower part of the intestine is involved constipation and tympanites are more pronounced and the tumor has more mobility, especially when located in the small intestine. If the *sigmoid* is invaded, edema of the left lower extremity may appear as the result of pressure, the stools will be scanty, offensive and mixed with pus and blood. When the *rectum* is involved pain, tenesmus, flattened stools and an abundance of mucus, pus and blood will be in evidence. A digital examination will establish the location of a morbid growth in the rectum. Tumors which have their origin in other organs must be excluded. A *floating kidney* may be suspected. This is more movable and may be returned to its normal location. It is often possible to outline the shape of the smooth kidney. Cachexia is not in evidence. A *movable spleen* should also be recognized by its characteristic shape, its smoothness, notching and its location. *Enlargement of the liver* may generally be traced by their continuity to the hepatic area. An *ovarian tumor* may be determined by joint vaginal and abdominal examination, to have its relation with the uterus. A *circumscribed peritoneal exudate* gives a flat percussion note and it is commonly associated with temperature. A simple inflammatory *stenosis* of the bowel is a condition which offers many confusing points in differentiation. Cachexia, however, is not present in the early stages, and evidence of some other cause may be discovered.

Prognosis and Treatment.—An unfavorable prognosis must be given in the majority of these cases. *Operation* according to Tuttle resulted in 14.8 per cent of cures with no return under three years. The principles of treatment recommended under intestinal obstruction may be applied so far as they are serviceable. The *physician's work* consists in sustaining the strength by the use of nourishing and assimilable foods and the relief of the constipation so far as possible. In the later stages it is a merciful procedure to relieve the patient with morphin.

HEMORRHOIDS.

Inasmuch as it frequently falls to the lot of a physician to undertake the treatment of hemorrhoids or piles, it seems best to discuss the subject so far as it pertains to our practice. This affection properly belongs to the care of the surgeon, except in the incipient stage when it is possible to give relief and perhaps perform a cure through the aid of internal medicine, or local treatments. The primary disease, whether it is located in the liver, the heart or in the intestinal tract, demands systematic treatment on the part of the physician. The cause of hemorrhoids rarely starts in the rectum.

Treatment.—It is not necessary to take up the features of etiology and pathology which characterize the disease, as those are explicitly enumerated in surgical works. Our practice deals with the treatment alone. *Constipation* is the principal perversion which we are to correct and the treatment of this has been outlined under a separate chapter. In the *early stage of hemorrhoids* it may be best to use a simple enema, in order to facilitate the movement without any rectal straining. Glycerin and wheat gluten suppositories may accomplish this result, or it is possible to get a free evacuation with only a low enema. Cathartics which irritate the rectum will cause intense tenesmus and smarting. Salines, in small but frequently repeated doses, will accomplish an evacuation with the least rectal pain, and I know of no better remedy for hepatic relief, without intestinal excitement, than *chinoidin*. The continued use of *iris versicolor, chelidonium, chionanthus* or *carduus,* in the tincture, will liberate the bile so that portal stasis is very soon overcome. These objects should not be sought by the aid of violent catharsis. Olive oil, or the more drastic castor oil, may often be called into use in severe cases, and it is possible that calomel will be demanded in some cases. The flaxseed tea, previously advised, is not only a relief to the constipation, but it is demulcent as well. The food should be highly nutritious, easily digested and have little residue.

The *local treatment* is a palliation which is absolutely necessary for temporary relief in acute cases and it may be a required procedure in chronic conditions. Rectal constriction is relieved by the careful stretching of the sphincters. This may be done by the finger well lubricated. The speculum or the dilator is too harsh and the information regarding the sphincteric relaxation which is conveyed to the finger makes this the instrument par excellence. This act relieves the constricted local circulation, and greatly reduces the tension and pain. At the same time it favors the fecal evacuation and minimizes the pain during the act. I am confident that I have cured many hemorrhoids by this method in the early stages. In the chronic stage we may also gain something by rectal dilatation and the local introduction of medication. The pile pipe or the speculum may be necessary as a means to this end. Esculus cerate, pine tar cerate, aristol cerate, hamamelis cerate and many other mild astringents may be thus introduced for local relief. Injections of rhatany, in mild solution, or hamamelis may act both as astringents and soothing agents. A favorite prescription of mine is a suppository containing rhatany two grains, hamamelis, two grains, and eucalyptus, one grain. This may be modified in accordance with the degree of local inflammation. Opium suppositories, one-quarter grain, may be required when the pain is extreme, and applications of ice are often necessary for this relief and for the reduction of the pile.

Internal remedies in acute seizures of this trouble are of more value than ordinarily realized. First in the list we should mention *sulphur* and *aloes*. The former, in its action upon the portal system through the liver, gives inestimable relief. Its typical symptoms which refer to the rectal disorder may be defined by the frequent and ineffectual desire for stool which naturally keeps the sphincters in a state of irritability. It moreover is demanded by the diarrheic excitement, the oozing from the rectum and the anal itching. I employ the remedy in the third or sixth decimal potency and I continue its use for some time. *Aloes* refers to a rectal incontinence and a high degree of inflammation, with a hemorrhoidal protrusion which simulates a bunch of grapes. It relieves the portal congestion if given in sufficient dosage and, therefore, overcomes the local distention of the veins in the rectal region. I give the second or third decimal potency, though it is claimed by others that the results are equally satisfactory when a higher potency is used. *Esculus, collinsonia, hamamelis* and *nux vomica* are four other remedies worthy of careful study in these cases. *Esculus* is indicated when a marked dryness of the mucous membrane gives rise to a feeling of splinters or sticks in the rectum. Prolapsus and constipation, from inactivity of the lower bowel, are other prominent symptoms, when it is demanded. *Collinsonia* is a remedy which is required when diarrhea alternates with constipation and the discharge is bloody or even dysenteric in character. We find, in connection with its symptomatic suggestions, positive evidence of cardiac insufficiency. Portal congestion is also so pronounced that varicose veins and edema or dropsy are recognized. *Digi-*

talis, convallaria, adonis, apocynum and *crategus* have a physiologic similarity to collinsonia. *Elaterium* may be considered under this line of symptomatology, but it has more definite relation with gastro-intestinal perversions. In one-eighth or one-tenth grain doses it may be used when there is need of prompt evacuation.

Hamamelis, in its internal action, is useful, both as a hemostatic—in heroic doses—and as a relief of local inflammation in potency. *Nux vomica* goes back to the digestive disturbances and in its action upon the unstriped muscular fibres it aids in helping the bleeding pile, and the derangement of the colonic mucous membrane as well. It is also the remedy to employ when the cause of hemorrhoids is due to alcoholic and drug excess, or to dietetic errors. *Bryonia,* with its dry and hard stools, should be compared with nux. *Dioscorea, hydrastis, bismuth* and *magnesia* may also be suggested in this connection. *Petroleum* is applicable when the rectal burning and itching are marked and the hard stools are covered with mucus. *Graphites* has a degree of usefulness when the rectal prolapse is complicated by anal fissures and the proctitis has extended outward until the surrounding tissues become excoriated or covered with herpetic eruptions. *Cina, alum, chamomilla* and *sulphur* may be compared in this respect. *Podophyllum* is somewhat useful in hemorrhoidal conditions when the cause arises from the irritation of a watery diarrhea. It is suggested by the yellow and offensive stool which contains mucus and undigested feces. Intestinal colic is also another characteristic symptom when this remedy is called for.

Phytolacca is of some value in the treatment of hemorrhoids, particularly when the bleeding is attended by neuralgic pains in the rectum. *Nitric acid* may be used when there is a low grade inflammation or the bleeding is the result of a degenerative process. *Pulsatilla* has an action upon hemorrhoidal conditions similar to that of *ipecac, mercurius* and *sulphur.* Aside from its typical symptoms we shall find the pressure symptoms, the pain, the itching and the watery diarrhea, all of which are characteristic indications in remedies of this kind. *Thuja* has been recommended on account of the condylomatous appearance which the chronic hemorrhoidal tumors may assume. It may be used as an internal remedy or applied locally. Other remedies may be studied in accordance with their symptomatic suggestions.

NEUROSES OF THE INTESTINES.

SECRETORY DISTURBANCES.

Mucous Colic.—This is an affection of the large intestine which is characterized by an excessive secretion of mucus. The mucosa does not manifest distinct pathologic changes, as the inflammatory process is not usually associated, though some claim that this is possible. As a neurosis it represents only an increase of catarrhal exudate from functional excitement. The majority of cases are found in nervous women and mechanical irritation, like horseback riding is supposed to excite the condition.

Symptoms.—At various intervals the stools will contain an *abundance of mucus,* which is usually ribbon-like in character; in rare instances casts have been expelled and the ejecta contains much turbid substance. *Colicky pains* and *tenesmus* usually prevail and obstinate constipation often exists. The symptoms have a tendency to appear in paroxysms which in some cases may last a week or longer. It may assume a chronic course if the nervous cause continues. The *diagnosis* will be made by the microscopic examination of the ejecta plus the symptoms outlined. The *prognosis* is usually favorable.

SENSORY DISTURBANCES.

Enteralgia.—This is a neuralgia of the intestines due to some reflex neurosis which may arise in connection with hysteria, the neurasthenic state or as the result of some direct nerve irritation. The etiology naturally goes back to such conditions as irritation from pelvic causes, or the excitement of nerve filaments when the intestinal mucosa is disturbed by foreign bodies like gallstones, enteroliths and gaseous distentions. Oxaluria may also be regarded as a factor. Local congestions which are due to the sudden attacks of cold, gouty conditions or motor excitement of the intestinal muscles which produce colic may also be reckoned as etiologic factors. Nerve shock or any sudden excitement may be prolific causes of this trouble.

Symptoms.—A sudden attack of *intestinal colic* is the typical description of enteralgia. Some cases develop slowly and gaseous eructations, flatus and abdominal tension precede the paroxysm of pain. When the painful sensations reach a crisis, the patient will assume a doubled-up position which is so characteristic of true colic. The pain is deep-seated, boring, tearing or cutting in character. It may be circumscribed or diffuse and the attack will subside as suddenly as it came or by degrees. A recurrence is common in the majority of cases. A form of enteralgia, in which the hemorrhoidal nerve plexus is involved, is termed *hypogastric neuralgia,* and we may find this in hysterical patients; it is also an occasional symptom of tabes dorsalis. The symptomatology is characterized by a distressing pressure sensation in the rectum and bladder, while the urination and defecation are performed with difficulty, despite frequent urging. The pain will radiate down the thighs, simulating, to a great extent, the acute form of sciatica.

Diagnosis.—*Neurotic enteralgia* must be differentiated from the colic which arises from some organic disease. In the latter, aside from the pathologic perversion, we shall find, invariably, fever, vomiting, constipation or diarrhea and increase of pain from the slightest pressure. *Renal colic* has pain which starts in the lumbar region and is reflected down the course of the ureters; the presence of blood in the urine will be a very suggestive symptom. Hepatic colic will be defined by jaundice, other symptoms of liver disturbance, and also by the location of the pain. *Myalgia* and the pains which are often called rheumatic will be distinguished by their individual peculiarities and the causes which develop them.

Decreased intestinal sensibility is recognized by the constipation which is caused by diminished peristalsis. The anesthesia may reach such a degree that the desire for stool is not appreciated and the accumulation of feces in the rectum may be enormous. In reality this is a symptom which attends brain or cord diseases and is a possible sequence following a prolonged or exhausting attack of nervous prostration. The intestinal atony is liable to reach a stage in which mechanical removal of the feces is necessary.

DISTURBANCES OF MOTILITY.

Nervous diarrhea may follow from the excitement of the motor nerves supplying the intestinal muscles. This may be due to reflex irritation or it may arise from disease of the central nervous system. Neurasthenia, hysteria and other forms of mental disturbance must be counted as predisposing factors, and it is an occurrence which may be noticed in patients who are endowed with highly sensitive nervous systems. Emotional excitement is often a cause.

Symptoms.—The stools are frequent, though not always of diarrheic character. Neither mucus, blood or pus are found in the feces. In fact, there is nothing in the character of the dejecta to indicate any disease of the intestine. During any excitement of the nervous system the stools are copious and frequent and these evacuations occur most frequently during the early morning hours. With all of this apparent waste there will be slight evidence of nutritional impairment. Organic diseases of the bowel will be easily eliminated by careful study.

Enterospasm occurs as the result of a spasm of both the longitudinal and circular fibres of the intestine. It may be so complete as to temporarily simulate bowel obstruction. In other instances the spasm may be only sufficient to create a compression of the feces so that they assume a ribbon-like form. The rectum also may be affected, with the result that a proctospasm interrupts the expulsive act sufficiently to obstruct the free passage of feces. The causes observed under enteralgia are the provocative influences and the diagnosis must exclude organic diseases which give similar symptoms. We must also differentiate the constipation which may be traced to a more direct neurosis or a paralysis from central lesions.

Treatment.—In the treatment of intestinal neuroses the *nervous derangement* will require first attention and the treatment in this respect will conform to that already outlined in separate chapters. *Dietetic corrections* will often be required and the patient must be treated in accordance with the accepted general management of the neurasthenic state. In the secretory neuroses some help may be obtained by the local use of enemas. The normal saline, with a few drops of peppermint added, may give temporary relief, when colic and gaseous distention are distressing. Remedies, and possibly mild cholagogues, which restore a normal amount of bile to the intestine, will not only overcome the abnormal secretions, but, by removing a possible irritating fecal mass, will relieve the intestinal colic. In rare cases it may be necessary to resort to opiates. These will be required mostly for the enteralgia or the hypogastric neuralgia. A rectal suppository containing one-fourth grain of opium is the best method for this purpose. Codein, internally administered in one-eighth or one-quarter grain doses, is the best antispasmodic remedy. In many cases a hypnotic will be more serviceable than an opiate.

During an attack of enteralgia some relief may be obtained by hot applications to the abdomen or a hot bath. *Colocynth* will often be indicated and when it is given at sufficiently frequent intervals, it may abort the worst spasms. *Dioscorea, belladonna* and *chamomilla* are often indicated and the sedative influence of *camphor, veratrum alb.* and *cuprum ars.* may be demanded. In a certain class of neurotic cases we shall find classical indications for *nux vomica, ignatia, physostigma* and *conium.*

Gelsemium, cimicifuga, pulsatilla and *caulophyllum* will meet another train of symptoms in neurasthenic and hysteric females, while such remedies as *plumbum aceticum, phos. acid, kali phos.* and *zinc pic.,* are indicated in a debilitated condition of the nervous system. *Alumina* should be prescribed when symptoms resembling lead colic appear, as in many cases we shall find this simulation. *Platina* may also be compared in this respect. *Cinchona* is efficient when malaria, sexual excesses or other debilitating factors are fundamental. *Coffea, hyoscyamus, agaricin* and *anacardium* will cover the more pronounced excitement of neurasthenia and hysteria, while *magnesia phos., silicea* and *carbo veg.* will often relieve the colic which is due to flatus.

The *complications* and *sequelae* are quite numerous. *Obstruction of the bowel* often exists as a result of a constriction produced by the adhesions formed by the attending peritonitis. *Hepatic abscess* is an occasional complication which follows a pylephlebitis due to thrombosis or embolism. The diaphragm may be perforated to the extent that pleural empyema and pyopericardium result. It is also possible that the brain and other tissues may be the seat of metastatic abscesses. Fecal, vesical and umbilical fistulae are not uncommon results when the abscess has penetrated these parts. Appendicitis may involve a hernial sac and fatal hemorrhage has occurred from the necrosis of an artery.

APPENDICITIS.

This term, which refers to inflammation of the appendix, also covers those terms formerly much used, typhlitis and perityphlitis. The latter two refer to inflammation of the cecum, but we now know that it is the appendix, and only rarely the cecum alone, which is involved.

Anatomy.—The appendix is attached to the cecum and "is a functionless relic of a large ancestral cecum." It is normally about one-fourth of an inch in diameter and varies in length from one to nine inches. Lying in the right iliac fossa it may extend downward over the pelvic brim, point toward the spleen or extend upward toward the liver. behind the ascending colon. These are the most common positions in which it is found, but it may be discovered in other regions. It is composed, from within outward, of a mucous, submucous, muscular and peritoneal coat and contains much lymphoid tissue. Its opening into the cecum is guarded by a fold of mucous membrane known as the valve of Gerlach. It has a short mesentery coming from that of the ileum between the layers of which runs a small artery—the appendicular. The inflammation probably always begins in the appendicular mucous membrane.

Etiology.—Primarily appendicitis is due to bacterial invasion of its mucosa, the most common micro-organism being the bacillus coli communis, and less commonly and usually associated with the former the streptococcus, staphylococcus and proteus vulgaris. Also the bacillus of typhoid and that of influenza may be the invading micro-organism. The *predisposing factors* are fecal concretions and foreign bodies, the former being very common, the latter much less frequently found, in one series of 1,400 cases reported by J. F. Mitchell being only 7 per cent.

The *anatomy* of the appendix also predisposes to inflammation of that organ. The outlet from it, guarded by the valve of Gerlach, favors retention of fecal matter, foreign bodies and bacteria, thus favoring irritation and the development of the bacteria within its cavity. *Constipation* and errors in diet are therefore important factors predisposing to appendicitis. *Violent exertion* may cause irritation of the appendix. *Age* is also a prominent factor, young adults being most frequently afflicted with this disease. *Sex* likewise plays a part in appendicitis, men being affected oftener than women in the ratio of six to one. One attack of appendicitis greatly predisposes to subsequent attacks.

Pathology.—The following forms of appendicitis are differentiated by the pathology rather than by the symptomatology: (1) Catarrhal appendicitis; (2) suppurative appendicitis; (3) obliterating appendicitis.

Catarrhal appendicitis is characterized by inflammation of the mucosa, which becomes swollen, narrowing the lumen of the tube. The mucosa is not destroyed. *Suppurative appendicitis* refers to a destructive inflammation of the mucosa with the development of pus. Should this process result in such destruction that the lumen be destroyed and obliterated, it is called *obliterating appendicitis.* If the peritoneal covering is involved in the inflammatory invasion it is known as *periappendicitis.* If abscess results outside of the appendix it is designated *periappendicular abscess.* The first three forms are differentiated by the degree of inflammation and pathologic change, rather than by the symptomatology.

Symptoms.—These we shall study in the order of their importance, constancy and sequence.

Pain. This is usually the first complaint of a patient suffering with appendicitis and should always lead to careful study of the case. At first somewhat diffuse over the abdomen, and often described as "belly-ache," it later becomes most marked in the right inguinal region. It is colicky and intermittent in character. Very often the pain is most intense in the epigastric region and has often led to a diagnosis of gastritis, biliary colic and renal colic. If, however, palpation is made over the appendix the real origin of the pain becomes apparent. In some cases the pain is referred to the groin, the thigh and the testicles, giving rise to confusion in diagnosis. *Sudden cessation* of the pain is of serious import and signifies rupture of the appendix, beginning gangrene of that organ, rupture of a periappendicular abscess or beginning generalized peritonitis. The degree of pain bears no relation to the degree of involvement of the appendix; many cases with trifling pain have, at operation, been found to have a suppurating or gangrenous appendix. For this reason one should never make light of a case of appendicitis.

Nausea and Vomiting. These are almost as constant symptoms of appendicitis as is pain. Nausea usually comes on early and lasts only a few hours. Pressure over the appendix may develop it. Vomiting at first depends upon the amount of gastric contents, and is sudden and forcible; should perforation and peritonitis occur it is more of a regurgitation and stercoraceous in character.

Fever. This may develop shortly after the onset of the disease and varies in character. Like pain, it is not a reliable indicator of the severity of the attack; it ranges from 99° to 104° F. A sudden drop in temperature with an increase in the pulse rate usually indicates perforation. After the onset the temperature curve is of the septic type. The *pulse* increases in proportion to the temperature. When perforation and peritonitis develop it becomes rapid, thready and of ominous import.

Tenderness and Rigidity. Soon after the onset tenderness and rigidity, the latter determined by palpation, are well marked signs. The former is often so marked that the patient can not tolerate the weight of the bed clothes on the abdomen. At first diffuse it later becomes localized over the appendix and is a very valuable sign. Rigidity is also at first somewhat diffused, but later is much increased in the right rectus abdominis. The whole abdomen is at first tympanitic and hard, later this is most noticeable over the lower half of the right rectus. Commonly the patient lies with the right thigh flexed upon the abdomen, in order to relax the tension of the rectus and thus gain some relief from pain. These signs are valuable in making the diagnosis. In connection with the foregoing symptoms *frequency of micturition* is often noted in this disease.

Physical Signs.—*Inspection* reveals much of value in appendicitis. The patient's face is usually pallid, anxious and expressive of intense suffering. We may also note the distended abdomen and the flexion of the thigh on the abdomen. *Palpation* confirms the tympany noted by inspection and discloses the rigid right rectus abdominis and tenderness. If the appendix is greatly distended and near the surface it may be palpated; if an abscess has formed it may also be recognized by palpation. To properly practice this the warmed hand should be gently laid upon the abdomen away from the appendix, and, the patient's attention being diverted from this procedure, the whole palm gently pressed over the different regions, the fingers making deep pressure to recognize the various organs. The right hand should be used in palpating the left side and the left hand on the right side. *Percussion* discloses also the tenderness, tympany and by a dull note the presence of any periappendicular abscess.

Leukocytosis is often present in appendicitis, the number of white cells varying from 16,000 to 25,000. Cases are met with in which the blood-findings are negative. When leukocytosis is present it usually indicates the severity of the infection and is generally an unfavorable sign. Its *absence,* however, does not necessarily signify a favorable outcome, as this may indicate a loss of recuperative power. The presence of leukocytosis is of value in differentiating appendicitis from typhoid fever.

Diagnosis.—In typical cases the diagnosis will not be difficult. A careful study of the symptoms described and their relation is of great value in making the diagnosis. *Sudden pain, nausea* and *vomiting, localized tenderness* and *rigidity* of the right rectus characterize the average case. A *tumor* near McBurney's point is quite often an early or a developing symptom. The difficulty of decision will come when these features are not clearly defined, which is not an uncommon occurrence. Inasmuch, however, as the base of the appendix is fixed it is generally possible to discover local tenderness at or very near the McBurney point. A most careful examination is required in each case and for that reason an anesthetic will be of great assistance in overcoming the rigidity of the abdominal wall. Digital examination through the rectum or vagina will often give valuable information. The fever, vomiting, constipation or diarrhea will help to confirm the decision.

Differential diagnosis must be exercised with great care. *Intestinal obstruction* will cause the most frequent confusion. Its characteristic symptoms, if studied carefully, will show a decided difference. Fever will not be a symptom unless peritonitis takes place; the pain is more intermittent and less liable to be located in the right iliac fossa. The constipation is complete and flatus is not passed. The vomiting is persistent and becomes stercoraceous. Shock and collapse often characterize the early symptoms, and the abdominal distention is quite general. *Intussusception* may be found in younger adults and children; bloody bowel discharges are common and a tumor may be felt by abdominal and rectal examination. *Malignant growths,* with the exception of cecal cancer, are usually located on the left side and display a slower course of development. A careful consideration of the symptoms and characteristics of intestinal obstruction should be entertained in order to make a safe differentiation. *Gall-stone colic,* though presenting diffuse abdominal pain at first, will later give rise to a paroxysmal pain with greatest intensity in the vicinity of the gall-bladder; it may later be reflected toward the right shoulder. In this disease rigidity of the upper half of the right rectus is in contrast with that of the lower half in appendicitis. Signs of hepatic disturbance will be detected, and in case of distention a superficial, mobile and pear-shaped tumor may be discovered in the region of the gall-bladder. Confusion will exist when the appendiceal tumor extends upward to a region near the gall-bladder.

Digestive disturbances may be mistaken for appendicitis, though the greater error occurs when the latter is mistaken for the former. In these instances the vomiting is often a deceptive symptom but we shall rarely discover fixed pain, tenderness or a tumor in the appendicular region. Enterocolitis will have the additional symptom of diarrhea and mucous stools which are not frequent features of appendicitis. The simulating signs of ptomaine poisoning may generally be traced to some dietetic error.

Renal colic is defined by a pain which radiates from the lumbar region to the groin and testicle. Hematuria is generally present but fever is not a common symptom. A *floating kidney* presents a movable tumor and relief is obtained when it is returned to its normal position. *Pyonephrosis* is detected by renal tenderness and the presence of pus in the urine. *Paranephritic* abscess can not always be differentiated; it is characterized by a pain similar to renal colic, though it may be occasioned by a suppurating perityphlitis when the inflamed appendix is located posterior to the colon. An exploratory incision will be necessary to confirm a diagnosis.

Lobar pneumonia sometimes presents symptoms similar to those of acute appendicitis and will be differentiated by carefully studying the case and especially by a thorough physical and sputum examination. As the case progresses the differentiation becomes more evident.

Pelvic diseases of women will often be confused with appendicitis, particularly when the appendix is located in the pelvic fossa. Right ovaritis will present many simulating symptoms but the tenderness and pain are less severe and uterine disturbances are always present. A suppurating ovarian cyst has a gradual onset, and the pain is of a dull character. Pelvic abscess may be detected by vaginal examination.

Extra-uterine pregnancy presents a history of menstrual irregularity and there may be decidual fragments in the hemorrhage. Should rupture occur it will be attended by marked collapse on account of the hemorrhage and shock. The temperature then is subnormal and the local findings will be located lower in the pelvic region. *Tuberculous peritonitis* will be differentiated by the gradual development of the pain, tenderness and fever, ascites and the absence of a right inguinal tumor. It is more gradual in its onset and the appearance of the patient indicates the profound toxemia.

Typhoid fever occasionally resembles appendicitis when the pain in the latter is not excessive and diarrhea attends. The right iliac tenderness and the tympany are certainly confusing but the gradual development of the symptoms, the temperature curve, the rose spots, the peculiar delirium and Widal's test should settle this doubt. In case of early perforation the difficulty of diagnosis will be increased. *Appendicular colic* or neuralgia of the right iliac fossa is often a confusing condition but the inflammatory symptoms of appendicitis are not present. The pain in these cases has been ascribed to a peristaltic contraction of the appendix.

Prognosis.—It is not possible to look upon this disease except with concern. The doubt in diagnosis, and the delay in heroic treatment, make the prognosis decidedly unfavorable. Everything depends upon the pathologic condition. Suppuration is always dangerous and perforation is attended with high mortality. If surgical aid is promptly summoned and if a perfect operation is performed, the large majority of cases recover. The improvement in surgical technique has made the operative procedure a safe undertaking.

Chronic appendicitis is liable to be the sequela of an acute attack and it is a constant menace to the patient. It may be manifest through a sense of discomfort in the cecal region or it may partake of a more general disturbance of the intestinal tract. Fecal impaction may result from peristaltic impairment. The pain is subacute in character and may, at times, show signs of exacerbation which resemble somewhat the acute attack. Fever is rarely high but the depreciating effects are shown in the gradual loss of nutrition and the failing general health. The patient becomes emaciated, neurasthenic and hypochondriacal. Many patients with chronic gastric derangement really have chronic appendicitis.

Treatment.—It seems almost unnecessary to speak of the medical treatment of this disease for in that respect we have no known method which we may consider safe or sure. It is per se a surgical disease and demands the attention and service of a competent surgeon from the very incipiency. For that reason any extended discussion in this respect is uncalled for in a medical treatise. It is as important to know when to operate as how to operate, and any interference by a non-experienced surgeon will endanger the life of the patient. Palliation should not be encouraged for the greatest danger is in delay. Too many practicians, having a mortal fear of operation, and placing too much faith in their remedies, permit the case to reach a stage in which operative interference fails to save life. It is this neglect which causes so many unfavorable operations, and more than this it leads to unfortunate discussions regarding operative procedure. When the surgeon is called too late it naturally creates an unfavorable opinion among the laity regarding the surgical possibilities.

It will be necessary, however, for the physician to be competent in making a diagnosis and to have a thorough knowledge as to the management of the case while waiting for the surgeon. This will comprehend a perfect understanding as to the use of remedies and other adjuvants in inflammatory conditions. It is claimed by good authorities that it is safe to defer an operation in the first attack when the cardinal symptoms subside in the course of twenty-four or forty-eight hours. The wise physician, however, will leave this decision to the competent surgeon, who certainly will not operate with undue haste unless the emergency demands it. The unfortunate tendency of some physicians to resort to cathartics or abdominal manipulation—thinking it may be a case of fecal impaction—deserves the severest censure.

The *first requirement* is absolute rest in bed, and this means a complete rest for the inflamed part. To accomplish this both the mind and body of the patient must assume a state of absolute repose. To obtain complete rest for the intestines food should be absolutely suspended until the inflammatory danger is passed. That there are non-operative cases we can not deny, but it is my contention that the surgeon alone is competent to decide as to these exceptions. In this respect I refer to the surgeon who is both capable and conscientious. *Opium* is a remedy which not only relieves pain but secures a certain amount of intestinal rest. It may have a sphere of usefulness in appendicitis, but its greatest danger is due to the fact that it may mask the cardinal symptoms and at the same time create unfavorable and annoying reactions, in peristaltic activity. For that reason it is doubtful if it has any place in the treatment of this disease.

The best thesis which covers this question of intestinal rest particularly referring to dietetic conditions has been written by Dr. A. J. Ochsner. I take the liberty in making free quotations in the following paragraphs: "First, the anatomical location of the appendix is such as to make its elimination from the general abdominal cavity comparatively easy, provided the surrounding structures remain at rest for a time." "Second, under a condition of rest the most important physiologic function of the surrounding structures (the cecum, the omentum and the small intestines) consists in surrounding any diseased or injured organ. In case the appendix is in a condition of inflammation the surrounding structures will at once crowd about this organ and form a perfect barrier against the introduction of septic material into the general peritoneal cavity. It seems to be the especial physiological business of the omentum to stand guard over the various intra-abdominal organs, to surround them and to protect the other organs against infection in case of any such danger. In the same manner it seems to be the function of the small intestines to enforce the efforts of the omentum by applying themselves to the latter on every suitable occasion. The condition obtained in this manner is, of course, most favorable because if it is not disturbed the worst possible result will be a circumscribed abscess."

The author then goes on to speak of the physiologic perversion of this natural support in case food is taken into the stomach inasmuch as this excites a continuous peristaltic action of the small intestines. This of course overcomes the omental protection against infection of the general peritoneal cavity and favors the distribution of septic material. He argues that the patient naturally objects to taking food as it not only disturbs the stomach but excites peristaltic motion of the small intestine. Another disturbance from food introduction is experienced in the formation of gas in the stomach and intestine. This must be expelled through the esophagus or it must pass into the colon through the ileo-cecal valve and thus cause severe disturbance in the inflamed appendix. He, therefore, concludes that the only non-operative treatment must be of a character to secure absolute rest of intestinal function and action. This he regards as the only way we can aid nature to care for an inflamed part except to remove the offending cause of trouble. To accomplish this there must be a complete suspension of all food by the mouth. No exception is made to liquid or prepared foods. This is certainly a more natural method of reducing peristalsis than by opiates.

To overcome the patient's exhaustion the author advises the administration of non-irritating nutrient enemata every three to six hours and these should not exceed four ounces at a time. Water may be taken by the mouth but in case this causes gastric or intestinal excitement it must be given by enemata. He also advises gastric lavage in case there is any sign of stomach disturbance, fearing, of course, that this irritation may reach the intestine. It is apparent that we have no better suggestions than the above for cases in which, for any reason, operation is not permitted.

Under the subject of treatment it is probably permissible to use local measures to relieve pain. The ice bag, if properly suspended, will be a valuable agent in this respect. It is possible also to obtain a similar relief through the aid of leeches and mild counter-irritants. It will also require the service of a physician to watch the stage of convalescence as the return to a solid diet and normal activity is a critical undertaking.

Internal remedies will always be of service and must be employed in accordance with the indications, but we must not rely upon these alone. *Belladonna, aconite, bryonia, baptisia, echinacea* and *rhus tox* will often meet the febrile symptoms of the early stages. *Mercurius corr., hepar sulphur, silicea* and *arsenicum iodid* must be considered as the inflammatory exudate develops.

DISEASES OF THE LIVER AND BILE TRACT.

ABNORMALITIES IN SHAPE AND POSITION.

Altered Shape.—Malformations of the liver have a congenital cause; they may exist as the result of disease or they may be due to pressure. The congenital class is associated with other imperfections of physical formation and development. The abnormality which follows the course of a disease is the sequence of a pathologic change and those which are secondary to a prolonged pressure are dependent upon external conditions. An example of the latter class may be observed in the so-called "corset liver." In this the right lobe is often divided by a transverse furrow while the lower part is atrophied by the continued pressure. The fibrous increase may become so pronounced that the normal parenchyma is supplanted by connective tissue.

The change in structure may cause confusion in physical examination and the clinical symptoms are rarely suggestive. A sense of pressure or weight is some times a complaint and pain may be experienced in case the peritoneum is irritated. The obstruction to the exit of bile may cause reflex gastric disturbance or favor the formation of gall-stones. In case of venous stasis there may be temporary swelling of the isolated part of a lobe. It must be differentiated from a tumor of the liver.

Abnormality of Position.—Malpositions may be due to congenital or acquired causes. In case of transposed viscera the liver is found on the left side. When the diaphragm is imperfectly developed the liver may extend into the thoracic cavity and when the abdominal parietes are deficient it may protrude anteriorly. In the acquired malpositions it may be pushed upward by ascitic fluid or abdominal tumors; it may be crowded downward by right-sided pleuritic effusions, emphysema of the right lung or an intra-thoracic tumor, and may occupy the epigastric or umbilical region. The term "wandering liver" has been given to those cases in which the liver displacement exists as the result of lengthening of its suspensory ligament. It occurs most frequently in women after confinement, and is a possible congenital condition. It usually returns to its natural location when the recumbent position is assumed. Physical examination will reveal a movable tumor which conforms to the size and shape of the liver, while the normal site is tympanitic on percussion or else comparatively dull when other organs occupy the liver's position. It is liable to be confounded with carcinomatous diseases of the omentum or the pylorus, tumors of the ovary or kidney, dermoid cyst and hydro- or pyonephrosis.

The symptoms are often negative though they may simulate those of the constricted liver with the addition of slight jaundice and marked hepatic colic. The treatment will consist of mechanical measures to hold the liver in position.

JAUNDICE OR ICTERUS.

This term refers to a condition in which the tissues and secretions of the body are affected by a yellowish discoloration due to the presence of bile. It is properly a symptom and not a disease. It is due to the deposition of bile pigment in the skin; in rare instances it may be caused by the faulty elimination of hematin from disintegrated red blood corpuscles. When it is due to bile absorption we call it hepatogenous jaundice and when it comes from disintegrated blood we use the term hematogenous or toxemic jaundice.

The bile is reabsorbed when there is an obstruction to its exit. This occurs in case of occlusion of the hepatic or common ducts or when catarrhal accretions in the duodenum prevent the outflow of bile into the intestine. Inflammatory adhesions, morbid growths in surrounding tissues and organs, as well as fecal impactions may be the obstructive causes. We must also take into consideration the pathologic changes which take place in the liver itself: cirrhosis, hepatic carcinoma, abscess, hyatid cysts, and even persistent circulatory perversions may give rise to jaundice. These are the typical causes of the hepatogenous variety and if we accept the hematogenous theory we must go back to the blood disintegration to find the cause. This form of jaundice has been denied by many of the best students and particularly Stadelmann, who claims that this form of obstruction is caused by the plugging of the smaller bile ducts by "viscid bile or catarrhal secretion or possibly by compression of these ducts by swollen adjacent liver cells or by leukocytic infiltration of the interstitial tissue."

ICTERUS NEONATORUM.

Jaundice of the new-born is a common occurrence at birth. It is a harmless disturbance and usually disappears in a few days without medicinal or dietetic aid. A patulous ductus venosus, diminished pressure on the portal vessels from ligation of the umbilical vein or external pressure are enumerated as the probable causes. A graver form may exist as a result of congenital stricture or absence of the hepatic or common duct, congenital syphilitic hepatitis or septic phlebitis of the umbilical vein.

The symptoms of true icterus neonatorum will be defined by a greenish-yellow color resembling that of chlorosis. The mucous membranes are pale and the conjunctiva shows only a slight discoloration. The icterus appears on the second or third day, the pulse is feeble and is accompanied by a soft systolic murmur with a venous hum in the neck. The urine does not show the presence of bile pigment, the bowels are not disturbed and there is no evidence of illness.

SIMPLE CATARRHAL JAUNDICE.

Inflammation of the common duct is characterized by a jaundice which is the result of retention and absorption of the bile. The most frequent cause is the extension of a gastro-duodenal inflammation. Passive congestion of the liver, secondary to cardiac or renal disease, and infectious diseases like pneumonia, typhoid and malaria may be the exciting factors. Exposure to cold, improper food and such irritants as tea, coffee and alcohol are possible predisposing causes. To a certain extent neurasthenia, excitement and mental anxiety may induce it. It is also claimed to be of epidemic occurrence.

Pathology.—The liver on post mortem examination is found enlarged, distinctly yellow and drops of bile appear on section. The mucosa of the common duct is swollen and the duodenal end is occluded by a catarrhal exudate. The inflammation may extend into the cystic and hepatic ducts and the gall bladder is usually distended with bile. Biliary cirrhosis may follow a connective tissue increase in case the disease becomes chronic. Suppuration rarely takes place.

Symptoms.—*Gastro-intestinal* derangements, accompanied by a certain amount of malaise, generally precede the typical signs. The appetite is impaired, the tongue is coated and nausea and vomiting are quite common. *Icterus* will be noticed at first upon the face and neck, the conjunctivae are discolored and the yellow tint very soon covers the whole body. It changes to a greenish hue as the disease becomes chronic. *Itching* will be bothersome, urticaria and other eruptions may appear and, in the severe cases, ecchymosis or more profuse hemorrhage may take place in the skin or mucous membranes. Bile pigment will be detected in the *urine* and, in protracted cases, albumin with bile stained tube casts may be in evidence. Hyaline casts are found even in the milder attacks. The perspiration is bile stained, and will be noticed by the discolored linen.

The *bowels* are constipated and the stools have a characteristic clay color and an offensive odor. When irritating and decomposing substances remain in the intestine diarrhea may take the place of constipation. The *pulse* is slow and the temperature normal unless there is a fecal impaction or the retention causes autointoxication. In this case the temperature may be elevated one or two degrees. The *nervous phenomena* are often extreme and grave cerebral symptoms may appear should there be any destruction of the normal hepatic tissue. Usually headache and vertigo constitute the most troublesome symptoms and these are due to the toxic effect of the retained bile.

Physical examination reveals an increased area of hepatic dulness upon palpation and percussion. The liver will be felt below the free margin of the ribs and in some instances the gall-bladder may be palpated.

Diagnosis.—The diagnosis is made on the appearance of jaundice without marked pain or signs of emaciation which point to the organic diseases of the liver. Young adults are more frequently affected and a history of dietetic errors and gastro-intestinal disturbance usually precedes.

Prognosis.—Simple catarrhal jaundice in itself is not a dangerous disease. It is only as it is associated with an infective cause or an organic hepatic disease that we find reason to fear the outcome. It has a tendency to self-limitation and easily responds to the simplest remedial measures.

Treatment.—The aim in treatment should be to remove the catarrhal occlusion of the common duct and to correct the causes which predispose before the smaller ducts are affected. This will first comprehend a dietetic regulation which will relieve the catarrhal condition of the stomach and duodenum and at the same time will not irritate the liver. The *simplest form of food* must be used and this should be administered in small quantities at stated intervals. *Functional rest* is absolutely necessary for the inflamed and catarrhal condition of the stomach and duodenum. For a time this will be obtained only by the use of warm milk and water or the simplest broths. Fats, starches and stimulating foods must be avoided even during the stage of recovery. Vichy, Apollinaris, Seltzer and similar waters may be used in the plain or carbonated form. *Mild aperients* like Carlsbad, Pluto, Rubinat and Hunyadi may be employed in moderate doses. It is a mistake to resort to catharsis unless there is positive evidence of serious obstruction. For that reason calomel or any other cholagogue should not be used. Effervescent citrate of magnesia or phosphate of soda, teaspoonful doses in one-half glass of hot water, may be given with good success two or three times daily. Other aperient waters will act with equal effect. Warm dilute alkaline drinks may be given ad libitum when a larger saline dose has a tendency to disturb the bowels. Warm baths and large enemas of hot water often afford much adjuvant relief. The latter will have a tendency to overcome intestinal fermentation and we may further favor this object by the internal use of betanaphthol or the sulphocarbolates. Ox gall, in five or ten grain doses, after meals, gives more satisfactory results in this respect. To permit its passage through the stomach unchanged the pills should have a keratin coating.

Remedies should be administered for the relief of the gastric and the duodenal catarrh. One of the first remedies to think of is the *dilute hydrochloric acid* in ten drop doses in one-half glass of water after meals. This will rapidly correct the gastric indigestion and thus obviate the formation of organic acids which can not help disturbing the duodenal function. *Nux vomica* is frequently indicated for the gastric insufficiency which is evinced by the loss of appetite, headache, foul taste in the mouth, the bitter eructations, itching of the skin and the constipation. *Podophyllum* refers to the intestinal derangement which arises from the torpidity of the liver and the catarrhal disturbance of the duodenum. The stools are liable to be green and watery or else there is extreme constipation with the hard, dry and clay-colored stools. Fever, sweats and hepatic pains are relieved by this remedy. *Chelidonium, chionanthus, euonymin, carduus, myrica* and *iris* are to be thought of when there is hepatic insufficiency and jaundice with gastro-intestinal disturbances. These, to my mind, should be given in large doses of the tincture in order to start the bile flow. Exception may be made in case of children and in patients who show a tendency to respond to light doses of any medication.

Mercurius dulcis, in the third potency, will often prove to be an effective agent if it is continued for an extended time. *Cinchona,* or better, *chinoidin,* in two to four grain doses every three hours until relief is obtained, has a decided action upon the liver when it is in a state of mild congestion. It may be compared with *cedron* and *natrum sulph. Leptandrin* works well in conjunction with cinchona. It is characterized by the vomiting of bile, aching pains in the liver and profuse, black and fetid stools. *Bryonia* has more pronounced pain in the hepatic region, exhaustion is marked, the jaundice is attended by urticaria or bullae, thirst is extreme and the stools are hard and dry. *Myrica cerifera* has all of the symptoms of hepatic disturbance found under chelidonium and it has in addition more influence upon the gastric and duodenal catarrh. Its characteristic gastric symptom is a tenacious, thick and nauseous secretion which is raised from the stomach. In this respect it resembles *hydrastis,* but more particularly *kali bichromicum,* both of which remedies have a sphere of decisive action in this disease.

Such remedies as *chamomilla,* in the jaundice of young patients, *pulsatilla,* in nervous women with menstrual trouble, *sulphur,* in that type which refers to some dyscrasia, *sepia,* which is characterized by its liver spots and the yellow complexion, and many others which may present a perfect system complex will contribute to the relief of catarrhal jaundice, though their fundamental action may not affect the liver. In this respect we shall often get much relief from remedies like *antimonium crudum* which is indicated by the thick white coating on the tongue, inasmuch as that is a condition indicative of a gastric and duodenal catarrhal exudate. The same is true of *arsenicum* which, by its symptoms, refers to a depreciated condition of the gastric membrane, and to *calcarea fluorica,* which goes back to a tissue depreciation. In fact this disease will respond to many remedies which are not apparently hepatic remedies, because the trouble has a more definite relation to other disorders.

When the heart is at fault and the hepatic perversion is in the line of passive congestion, it will be necessary to resort to such remedies as *digitalis, strophanthus, convallaria,* and *adonis,* while *crotalus, naja* or *lachesis and phosphorus* may be studied with interest when a fatty metamorphosis of the liver is a pathologic condition.

ACUTE INFECTIOUS CHOLECYSTITIS.

Acute inflammation of the gall-bladder is caused by pathogenic bacteria. It is not a very common disease. The pathogenic bacteria which infest the small intestine, like the colon bacillus and the bacillus of typhoid fever, are the most frequent causes though the pneumococcus, staphylococcus and streptococcus are also occasional causes. Biliary calculus with its attendant inflammatory adhesions, is no doubt a predisposing factor which favors the inflammatory progress. It is a question whether the direct extension of inflammation will create the disease.

Pathology.—The pathologic conditions are recognized under three distinct varieties—*catarrhal, suppurative* and *phlegmonous*—all of which represent the different degrees of involvement. The gall-bladder, in extreme cases, will be filled with mucus, muco-pus or pus and, at times, the exudate may partake of the hemorrhagic character. The cystic duct may be occluded and perforation may succeed the ulceration or gangrenous process. A localized abscess or acute diffuse peritonitis are possible sequences. Adhesions may cause attachments between the gall-bladder and adjacent viscera.

Symptoms.—Sudden or paroxysmal abdominal *pain* characterizes the onset. It is generally located to the right of the median line or in the epigastrium. *Tenderness* may be discovered in the region of the gall-bladder and this may become more diffuse as peritonitis intervenes. *Fever,* frequently attended by a chill and followed by sweats, is often recognized, though this is by no means a regular symptom. *Jaundice* will be noticed when the hepatic or common duct is implicated. *Rigidity* of the upper half of the right rectus abdominis may be observed and in some instances it may be possible to discover a *tumor* in the vicinity of the gall-bladder. This may be confused with a circumscribed peritoneal abscess following perforation.

Nausea and paroxysmal vomiting are early and sometimes persistent symptoms. When fever is present the pulse is rapid, and prostration will be pronounced. The abdomen is usually distended. Some cases will be ushered in by an early perforation which generally leads to a sudden and fatal termination. A leukocytosis may be found in the suppurative forms.

Diagnosis.—If the physical examination together with the symptoms conforms to the features outlined above, a diagnosis may be positively made in many cases. Differentiation must be made in the case of *appendicitis* when it is located high in the abdominal cavity. *Acute intestinal obstruction* may simulate it when the adhesions affect the intestine. *Localized peritonitis* will show a similarity in many of its symptoms. It will be necessary to make a careful study relative to the possibility of *typhoid fever* with symptoms which show an associate affection of the gall-bladder.

Prognosis.—The mild cases which are characterized by a catarrhal inflammation will respond to medicinal measures. Should the inflammation take on the purulent or gangrenous form a fatal result may be expected. Much depends upon the promptness of surgical relief; perforation is always to be feared.

Treatment.—Operative procedure presents the most positive and safest hope and this must be undertaken early. Relief of pain may demand opiates, and remedies may be used in accordance with the indications in the catarrhal variety. Rectal alimentation will relieve the gastric and hepatic functions.

CALCULOUS CHOLECYSTITIS.

Calculous cholecystitis naturally refers to a condition of which biliary calculi or gall-stones are the chief causes. These concretions are formed in the gall-bladder in connection with chemical changes in the hepatic function and the process of infection.

Etiology.—The precipitation of cholesterin is the principal cause and this is aided by the thickened bile; it is combined with the lime salts, bacteria and epithelial debris. The micro-organisms, mentioned in the acute or infectious variety, are the exciting factors, and the cholate salts of sodium suffer a decomposition as the result of these agencies. There are also *predisposing causes* such as sex, since females are more inclined to the disease than males, and age, inasmuch as a majority of the cases occur among those of advanced years. A sedentary life, irregular meals and foods which have an abundance of starches and fats are also contributing influences. Constipation, tight lacing and pregnancy are believed to be causes. Catarrhal jaundice and tumors which interrupt the outflow of bile are also to be considered as predisposing factors. Nervous diseases exert an indirect etiological influence.

Pathology.—"The gall-stone itself is a brown object, nearly spherical, oval or faceted and even polygonal in shape, usually the size of a pea or as small as a millet seed, producing in aggregation 'gall sand.' The faceted shape is caused by close packing of a large number of stones in a gall-bladder, as frequently happens. More rarely the stone is irregular—mulberry shaped. In addition to cholesterin, which makes up from seventy to eighty per cent of most stones, they contain various but still small amounts of bile pigment, calcic carbonate and organic matter. On *section* the stone exhibits either a concentric or homogeneous appearance, with or without a nucleus of bile pigment or organic matter, and very rarely of some foreign body. The cholesterin stones are almost completely soluble in etherized alcohol whence beautiful crystals of cholesterin may be obtained after evaporation." (Tyson.)

The *accretions* may accumulate in the gall-bladder in great number. They may also occlude the common or cystic duct and under these circumstances these tracts are often greatly dilated. When the cystic duct is implicated it may suffer such a dilatation and be filled with such a large amount of colorless, viscid and albuminous fluid that it may be mistaken for an ovarian cyst. *Suppuration* and *ulceration* may lead to a perforation which will involve the peritoneal cavity and perhaps adjacent organs.

Symptoms.—*Acute impaction.*—Biliary colic is the typical symptom of acute impaction of the gall-bladder or ducts. It is caused by the lodging of calculi during their transit through the cystic or common duct. It occurs as an acute paroxysm and it is extremely severe in character. The epigastrium is the seat of most intense suffering and the pain generally radiates into the abdomen and upward toward the right shoulder. Abdominal rigidity is not uncommon on the right side. The paroxysm usually ceases when the stone escapes from the duct. Remissions of the symptoms may appear.

Nausea and *vomiting* are usually present. *Fever* is preceded by a chill. The *temperature* reaches 102° or 103° F. but it usually subsides when the stone is dislodged. It is sometimes possible to detect crepitus when the gall-bladder is crowded with calculi and the patient is thin. *Jaundice* will be in evidence when the stone occludes the common or hepatic duct long enough for absorption to take place. The liver is usually enlarged. *Collapse* is a common feature, particularly when the pain is extreme and when peritonitis intervenes.

Symptoms of Chronic Impacted Gall-Stones.—These will vary in accordance with the location of the calculus and the extent of the infective cause. Many of the symptoms of acute impaction will be observed. When the *cystic duct* is the seat of obstruction dilatation of the gall-bladder will be a prominent feature. This accumulation comes from the inflammatory secretion of the mucosa. As mentioned before it may assume large proportions and be mistaken for an ovarian cyst; it is often palpable below the free margin of the ribs. *Jaundice* is rarely present. When the obstruction is in the *common duct* the gall-bladder may suffer only moderate dilatation. In simple chronic cholangitis the common duct is dilated and this dilatation may involve the hepatic duct. *Jaundice* is present, *pain* is paroxysmal and often assumes an "ague-like" character in which chills and sweats are extreme. The *temperature* may reach 105° F. and may assume a septic type. *Nausea* and *vomiting* are attendant symptoms. The liver is slightly enlarged and the stools may be bile-stained at times, though they are generally clay-colored. The spleen is often enlarged.

Naunyn gives the following diagnostic symptoms of stones impacted in the common duct: (1) The continuous or occasional presence of bile in the feces; (2) distinct variations in the intensity of the jaundice; (3) distention of the gall-bladder varying in degree with the seat of obstruction; (4) enlargement of the spleen; (5) normal size or only slight enlargement of the liver; (6) absence of ascites; (7) presence of febrile disturbance; (8) duration of the jaundice for more than a year.

Suppurative cholangitis manifests a fever of the septic type. Jaundice is slight, the liver is tender and enlarged, the duration is shorter and a fatal termination may be expected. As the inflammation invades a greater portion of the liver and gall-bladder, hepatic abscess and empyema of the gall-bladder are common results.

There are *other forms* of impaction in which fistulae are frequent terminations. These may form communications between the gall-bladder and liver or they may perforate into the gastro-intestinal tract, the pleural cavity, the urinary and other organs. Atrophy of the gall-bladder is a frequent result in the chronic case, while suppurative phlebitis and abscess of the liver are not uncommon. At times the stone may be so large that it is discharged into the intestine by ulceration.

Diagnosis.—The diagnosis in acute or chronic impaction of the gall-bladder may be made after a careful study of the foregoing symptoms. The following differentiations must be made: *Gastralgia* usually presents some sign of neurotic disturbance. The paroxysmal pains are either confined to the epigastrium or are reflected directly through to the back. It may be relieved by heat and pressure often palliates the suffering. Fever is absent, jaundice is not observed, the stools show the presence of bile and contain no stones. The urine contains no appreciable bile and the hysterical nature of the paroxysm may be recognized. *Renal colic* starts in the lumbar region and is reflected down the course of the ureter into the scrotum or labia. Micturition is painful and frequent, while the urine is scanty and often mixed with blood. *Intestinal colic* presents a boring pain in the vicinity of the umbilicus. Abdominal distention is a disturbing symptom and relief is obtained by pressure and the passage of flatus. It can often be traced to errors in diet. *Lead colic* will be settled by the history, the blue line along the gums and the wrist drop. *Ovarian pains* will be located in the pelvic region and some history of uterine disorder will be discovered.

Treatment.—Our first concern should be to remove the cause and this at once comprehends preventive measures in anticipation of future attacks of the biliary colic. This should begin with *dietetic regulations.* Fatty foods, sugars, starches and pastries should be prohibited and a simple diet, with an abundance of vegetables, should be adhered to. An abundance of pure water should be used with the meals and the habit of free libation of this should be encouraged during the intervals between meals. A favorite method of mine is to put one or two teaspoonfuls of Carlsbad salts into one or two quarts of water and compel the patient to drink this in twelve or twenty-four hours. Systematic exercise should be undertaken. Remedies which relieve the febrile condition may be employed according to their indications. *Belladonna, bryonia, baptisia, gelsemium, eupatorium* are frequently useful in this respect. We shall also need the service of remedies the action of which tends to relieve the hepatic torpor. In this respect we shall be served by *chelidonium, chionanthus, carduus, iris* and *euonymin.* To obtain success these must be given in tincture and continued a long time. Other remedies like *berberis, hydrastis, myrica, laurocerasus* and *cinchona* will be called for when indicated. *Colocynth, cuprum, dioscorea* and *terebinth* often relieve the spasm while *ignatia, cannabis* and *agaricin* may correct the hysterical features when present. Alkaline waters and the mild aperients of this character will have a tendency to liquefy the bile and thus favor its passage into the duodenum. Sodium phosphate, sodium salicylate and sodium sulphate or the Carlsbad salts will be of inestimable service in this respect.

The management of *biliary colic* will constitute another phase of the treatment and here we are to resort to measures of palliation as well as cure. I doubt very much if this stage of the disease will respond to many of the remedies with the phenomenal results which some of our ardent homeopathic practicians claim. In fact I consider such statements unfair to our therapeutic principle as well as misleading to the student and honest practician. Biliary colic must be relieved, and it will generally require the use of *morphin* hypodermatically administered. Palliation may be obtained by inhalations of *chloroform* but it will not be lasting. Hot baths, counter irritation and hot fomentations will help but not entirely relieve the paroxysm. For biliary colic I have found *glonoin* (gr. 1/100) and *hyoscyamin* (gr. 1/250) repeated till effect, very serviceable. A smaller amount of morphin is frequently required with these. Hypodermics of *strychnia* may be needed to overcome the cardiac weakness which may follow a collapse. *Olive oil* in heroic doses may aid in the liquefaction and passage of the bile. Calomel and extreme cathartics should not be used. Remedies like *ipecac, iris, nux vomica, arsenicum* and other indicated remedies will relieve the nausea.

When it is necessary to remove the gall-stones the services of a surgeon will be demanded and this calls for no therapeutic explanation in a medical work.

CARCINOMA OF THE GALL-BLADDER AND BILE DUCTS.

Etiology and Pathology.—The gall-bladder not rarely suffers from carcinomatous development. It is claimed as a primary disease when it starts in the fundus, and it may extend from this point to the liver and stomach. Occasionally it occurs as the result of contagious invasion. Calculi no doubt act as exciting factors when the ducts are implicated. Age is a predisposing factor. The cicatricial tissue resulting from the ulcerative process, due to the presence of calculi, is a favorable nidus for its development.

The gall-bladder may be distended as a result of the cancerous occlusion of the ducts, and it may reach large proportions. It is filled with a cloudy liquid resembling barley water, which contains flakes of epithelium, granular matter and inspissated bile. The common and cystic ducts, and likewise the ramifications of the hepatic ducts, suffer distention when the growth involves the common duct. Hepatic enlargement, with here and there an isolated nodular spot, will complete the pathological picture. Frequently empyema of the gall-bladder is a complication.

Symptoms.—The signs of this disease are similar to those which are found in other cases of obstruction. *Tenderness,* paroxysmal pain, vomiting, a rise of temperature, bloody stools, and occasionally dropsy, are typical features. *Jaundice* will not be observed unless the hepatic or common duct is affected, and then it will be intense. Inasmuch as this pathologic condition generally exists, we may regard jaundice as a characteristic symptom. Associated with the above symptoms a *cancerous cachexia* and *general emaciation* will be invariably recognized. A hard and uneven *tumor* may frequently be palpated in the region of the gall-bladder, which is enlarged as the result of obstruction in the common duct. The *urine* contains bile pigment and bile-stained casts, while the feces do not show the presence of bile. Ascites may be a symptom when the enlargement invades the liver and surrounding tissues.

Diagnosis.—The signs of chronic obstruction with the intense and increasing jaundice (in the majority of cases), the cachexia and emaciation, the constant pain, the advanced years of the patient, the sex (women being more frequently affected) and the physical findings ought to leave no room for doubt.

Prognosis.—The result is the same as in all organic developments of the kind. Death is the inevitable termination. The *treatment* at best can only furnish palliation. The relief of pain is about all a physician can accomplish. Operative measures are not generally successful.

STENOSIS OF THE BILE DUCTS.

Stenosis of the common duct is an incomplete occlusion which may occasionally arise as the result of inflammatory adhesions or external compression. Foreign bodies, as seeds, ulcerations and cicatrization following the passage of gall-stones, aneurysmal enlargements of the abdominal aorta or of the celiac axis, enlargement of the lymphatics of the liver, together with the rare obstructions formed by parasites, are some of the causes to be considered.

Pathology.—When the stenosis is recent, the liver is enlarged, congested and somewhat increased by connective tissue hyperplasia. When the obstruction is of long standing, the connective tissue formation is excessive, the cells are atrophied and the size of the organ will be diminished.

Symptoms.—The symptoms do not differ materially from those of chronic obstruction by gall-stones. The paroxysmal pain, the increasing jaundice, the vomiting and the fever constitute the typical features by which we define the disease.

Diagnosis.—In making a diagnosis from the above symptoms it will be necessary to study carefully the etiology and the few physical signs present. The history of the causative disease will furnish much help in making the decision. This disease may naturally be confused with *carcinoma of the pylorus* or *head of the pancreas,* but the evidence furnished by palpation in these diseases, together with their rapid development, the absence of the cancerous cachexia in stenosis, and the other characteristic differential signs ought to make the diagnosis comparatively easy. *Aneurysm* of the abdominal aorta will be recognized on finding a throbbing tumor in the epigastrium with the characteristic expansile pulsation. It will not be difficult to make the differentiation from other simulating diseases.

Prognosis and Treatment.—The cause of the stenosis will decide the prognosis. Generally speaking, the disease does not offer a hopeful prognosis. When foreign bodies are the cause we may expect some relief from alkaline waters, cathartics and internal remedies. When gall-stones are present we may rely upon the medicinal and surgical means suggested in their treatment. When stenosis follows ulceration of the duct it is possible to get some relief from a cholecystenterostomy performed by a capable surgeon.

VASCULAR AFFECTIONS OF THE LIVER.

Perversions in the normal circulation of the liver deserve attention, inasmuch as they are affections which must be considered in the treatment of the diseases of this organ. *Anemia* of the liver does not furnish any positive symptoms, and yet it is a condition which attends the general anemic state; or it may be secondary to fatty or amyloid degenerations. *Active hyperemia,* or acute congestion, is an excess of arterial blood in the liver which is a physiologic sequence following a hearty meal, or it may be associated with inordinate eating or the constant indulgence in alcoholic beverages. It represents the acute stage of any inflammatory invasion of the organ and it, therefore, may attend any of the specific infections. *Traumatism* has been regarded as a cause and *vaso-motor irritation* following such conditions as menstrual suppression is frequently causative. *Gastro-intestinal catarrh* and the catarrhal affections of the bile ducts naturally predispose to hyperemia. The *pathologic features* are characterized only by the apparent swelling of the liver, the change to a darker color in its stroma and the evidence of a distended capsule. The symptoms of hepatic origin are not marked except for a slight fulness and distress in the hypochondriac region, which are transient.

PASSIVE HYPEREMIA.

This is an engorgement of the liver, due to obstruction of the hepatic veins from tumors, by thrombi or a diseased condition following syphilitic phlebitis. Tight lacing has been considered as a factor. Among the general causes we may mention the weak or dilated heart, uncompensated valvular lesions, pulmonary emphysema, cirrhosis and intra-thoracic tumors.

Pathology.—The liver is enlarged, smooth and tense; its capsule is distended and the color is changed to a deep red. The center of the lobules is deeply pigmented, while the periphery is lighter in color, owing to the tendency to fatty infiltration. This creates a mottled condition which has given rise to the term, "nutmeg liver." The hepatic structure is somewhat softened and the cut surface will show an excess of blood. Later changes will reveal a connective tissue increase as the round cells proliferate and atrophy affects the parenchyma. The capillaries become distended and, as a brownish pigment is deposited in the center of the lobules, the liver becomes very dark in color. This has been defined as the stage of red atrophy, and as the atrophy increases with the development of connective tissue it is termed the "atrophic nutmeg liver."

Symptoms.—It is not always possible to discover symptoms which refer directly to passive hyperemia. The suffering may appear to be in the stomach or intestine. A *sense of distress,* bordering upon pain, may be complained of in the right hypochondriac area. The *cardiac symptoms* may point to the predisposing general cause and hematemesis is not uncommon. *Slight jaundice,* occasional clay-colored stools and biliuria usually attend a protracted case. *Dropsy* may be evidenced by the presence of a generalized edema or ascites. Palpation will discover a tender and enlarged liver and hepatic pulsation is frequently observed; the spleen may show some similar enlargement. The urine is usually scanty.

Diagnosis.—It is not always possible to make a correct diagnosis, inasmuch as it is necessary to rely very much upon the discovery of its cause. The *prognosis* depends upon the cause, but it may usually be considered as favorable. The *treatment* should be directed to the removal of the conditions which favor its development, and this will be discussed under different subjects.

PYLETHROMBOSIS.

Thrombosis of the portal vein is not of common occurrence. It may be favored by pressure from without in the case of a neighboring tumor, proliferating peritonitis, abscesses or enlarged glands. It may invade the smaller branches of the vein in the course of hepatic cirrhosis or it is liable to form in the larger branches through cancerous proliferation or gall-stone occlusion. Traumatism is a supposed cause and splenic disease may favor it by the retarding of portal circulation.

Pathology.—The usual thrombotic changes will be observed. As the degeneration progresses the adherent clot undergoes fibrous development in conjunction with the affected blood-vessel; this is liable to cause an adhesive pylephlebitis. Collateral circulation is frequently established and may compensate the obstructed portal circulation.

Symptoms.—Symptoms which are suggestive of pylethrombosis are rarely seen except in association with hepatic cirrhosis. *Ascites* develops more rapidly than in hepatic cirrhosis and this often forms a diagnostic symptom. *Hemorrhage* may take place from the nose, stomach and intestines. *Jaundice* occurs when biliary obstruction follows and diarrhea is not uncommon. Palpation discloses an enlarged and tender liver, and the same is true of the spleen to a considerable extent. Percussion will confirm this and also establish the presence of ascites.

Diagnosis.—It is a difficult matter to make a positive diagnosis. The sudden appearance of the symptoms and the signs of portal engorgement are the most important facts to be ascertained. Should sepsis supervene abscesses will follow and hemorrhagic infarct is a possibility.

Prognosis.—The disease always terminates in death and this is liable to take place suddenly. There may be a temporary improvement, but it is not lasting. The treatment will conform to that of interstitial hepatitis, and in case of abscess formation surgical intervention may be required.

SUPPURATIVE PYLEPHLEBITIS.

This is a purulent inflammation of the portal vein or some of its branches. It most frequently follows appendicitis, though a general infective process may create it. We notice this mostly in the ulcerative pyemic conditions which exist in the gastric, intestinal or pelvic regions. Typhoid fever and dysentery may be the source of infection.

Pathology.—Thrombi obstruct the small branches of the portal vein, the connective tissue stroma is thickened, while the coats of the veins present minute ecchymotic spots. These thrombi throw off fibrinous clots which, being carried into the hepatic circulation, form metastatic abscesses. These may become confluent and form one large abscess. The suppurative emboli may enter other veins and in this way may infect other organs and tissues. The liver is uniformly enlarged and, on account of the suppurative process, has a mottled appearance.

Symptoms.—The *subjective symptoms* pertain to the liver, inasmuch as metastatic abscesses are liable to exist here as the result of the pylephlebitis. Should this not be the case a systemic sepsis will be the clinical picture. The liver being enlarged and inflamed is *tender* on palpation. *Pain* may or may not be a marked feature. When a large abscess exists the hepatic enlargement is marked and the pain and pressure will be consequently more intense. *Septic fever* is a characteristic expression of the disease and this is usually accompanied by rigors and profuse sweating. *Jaundice* is generally present and *diarrhea* is not uncommon. *Gastric disturbances* are evidenced, displayed by nausea and often by vomiting. These cases usually assume an asthenic type and terminate in coma and death.

Diagnosis and Prognosis.—The foregoing symptoms offer the only guidance in making a diagnosis, which is not an easy matter. The hepatic complications give the most positive suggestions in this respect. The disease is usually incurable, its duration not often exceeding two weeks. When the abscesses are small, surgical intervention gives much encouragement.

Treatment.—Relief of pain is the only objective in the medical treatment. Symptomatic treatment will apply for various conditions. Surgical aid should be called when there is a localized hepatic abscess.

FATTY LIVER.

This term is employed to define two forms of fatty change in the liver structure. One refers to the infiltration of the hepatic cells with fat drops which interrupt the nutritive function without destroying the cell integrity. The other is a metamorphosis of the cell-protoplasm into fat which not only impairs the cell function but ultimately ends in complete cell-disintegration.

FATTY INFILTRATION.

Etiology.—Fatty infiltration is often a result of obesity in which the liver is filled with an excessive amount of fat. It is due to an over ingestion of fat-producing foods or alcohol. It is also a sequence of the cachectic state when fat accumulates in the liver as the result of improper oxidization. Chronic diseases of the heart and lungs promote fatty infiltration, as they interfere with the supply of oxygen to the blood.

Pathology.—The liver is decidedly enlarged, smooth and pale, while the microscope will reveal the drops of fat in the hepatic cells. The liver pits upon pressure, its edges are rounded and it has a light yellow or a grayish color. The infiltration may appear in localized areas or it may affect the parenchyma as a whole. Portions of the liver substance will float in water.

Symptoms.—The symptoms are not distinctive, inasmuch as the function is not greatly impaired. The bile-forming function is not decidedly interfered with and hence there is no marked jaundice, though the stools are pale in color. Percussion reveals moderate and uniform enlargement. Anemia and debility are often prominent symptoms. It is not uncommon also to find a feeble and irregular pulse, which is indicative of cardiac arrythmia.

Diagnosis.—It is not a difficult matter to make the diagnosis, inasmuch as we invariably have the causative symptoms to direct us. The lack of signs which point to portal obstruction will differentiate from *fatty degeneration*. The spleen is not enlarged, albuminuria is not present, and the hepatic enlargement is not excessive, hence it can not be confounded with *amyloid degeneration*. The absence of persistent jaundice, enlarged spleen and ascites rules out *fibrous hepatitis*. The smooth and uniform enlargement, without any associate infectious signs, will contrast clearly with the *inflammatory* invasions of the liver.

Prognosis.—It is possible to give a favorable prognosis, inasmuch as the majority of cases show no decided impairment of the hepatic function. The cause of the disease is generally easily eradicated and unless there are some unfavorable complications we may expect a recovery.

Treatment.—The principal object of the treatment is to remove the provoking cause, and this can be accomplished in the average case by careful dieting, the promotion of the digestive functions and the taking of systematic exercise.

FATTY METAMORPHOSIS.

Etiology.—The conversion of the cell protoplasm into fat and the subsequent process of disintegration make this a disease of far more serious consequences than fatty infiltration. Alcoholic excess may have something to do with its development, but it more frequently follows the results of wasting diseases like carcinoma and tuberculosis. Phosphorus or arsenic poisoning, pernicious anemia, and acute infections or intoxications which deplete the oxygen supply of the blood are also causative factors. It is also associated with amyloid and atrophic changes.

Pathology.—When there is pronounced fatty degeneration the liver shows marked atrophy. Its substance is soft, pliable and easily torn, and the color is light yellow. On section we shall find the acini replaced by fat cells and oil globules, and areas of pigmentation will be detected. The microscope reveals a globular appearance of the cells. Granular debris, cholesterin, tyrosin and phosphatic crystals are also discovered in the degenerated tissue.

Symptoms.—As in fatty infiltration we are not, in the ordinary case, able to find any constant and positive symptoms of the degeneration. Pain is an uncommon complaint, jaundice is an exceptional symptom and ascites appears only occasionally. When there is an associate fatty change in the kidneys the urine is scanty and albuminous, while fatty casts and crystals of cholesterin, leucin and tyrosin may be easily detected. In such cases the heart becomes feeble in its action, syncope and vertigo often being observed as expressions of the gradual muscular degeneration. Edema and anasarca are natural symptoms in this extreme. Physical examination reveals the gradually increasing atrophy of the liver.

Diagnosis.—This is determined chiefly by careful elimination of other diseases. The history of alcoholism, drug poisoning or some severe infectious disease will be necessary to confirm the diagnosis. Then the grave symptoms such as albuminuria, edema, ascites and cardiac failure which appear when the kidneys are involved, together with the progressive atrophy, are about all the guiding points we can find on which to base the diagnosis.

Prognosis.—Much depends upon the degree of fatty degeneration. If it is caused by overeating and indulgence in stimulants some relief may be expected when these errors are corrected. If it is due to a wasting disease everything depends upon the course of that infection. In this class of cases we can not expect a recovery, but only to arrest the progress of the disease.

Treatment.—While the incipient case alone offers hope in treatment, it is often possible to accomplish more than is expected in this respect. *Dietetic* regulation is of great importance, particularly when the patient has been too indulgent. *Hygienic considerations* are also valuable, inasmuch as the general strength and eliminating powers must be constantly maintained. Systematic exercise will be an adjuvant in this respect. We must not expect our treatment to avail us much when the pathologic changes are well established or when they are due to a well established wasting disease. If, however, it is possible to check the progress of that disease we may have a commensurate hope of retarding the fatty degeneration of the liver. Remedies like *iron* and *arsenic,* which are of such value in anemic conditions, should have a sphere of usefulness in this disease. *Phosphorus* should always be studied when there is a fatty metamorphosis. In conjunction with this we may do well to look into the symptomatology bearing upon *calcarea carb., natrum sulph., silicea, sulphur* and similar remedies. Should a syphilitic cause be found we can not do better than to employ the *iodids* or *mercurius* in its various forms. *Aurum chlorid* should never be overlooked, inasmuch as it applies not only to a specific degeneration, but it answers also in the tissue depletion from other causes. It is of inestimable value when the nervous system is first affected, and when the interstitial changes are also developed in the heart, liver and kidneys. *Nitric acid, phos. acid* and *ferrum phos.* may be compared in this respect.

Remedies like *chelidonium, chionanthus, cholesterin, china* and *leptandrin* have their sphere of usefulness when acute hepatic symptoms appear, and to a certain extent they are efficient in the incipient stages of fatty degeneration. The pernicious anemia should be corrected by *quinin, arsenic, iron* and similar remedies, and to accomplish any results, in this respect, they must be given in material doses and continued for their physiologic effect. Cardiac weakness will demand special attention and the usual tonics, but *strychnia* particularly must not be overlooked. *Naja, crotalus* and *lachesis* claim some therapeutic attention. Other remedies may possibly be indicated for the relief of certain symptoms, if not for a cure.

THE AMYLOID LIVER.

Various definitions, such as lardaceous liver, waxy liver and albuminoid liver, have been employed to define this disease. The term used above has, however, received more unanimous consent, inasmuch as the disease represents a gradual amyloid infiltration of all the hepatic tissues. The degeneration manifests itself first in the walls of the branches of the hepatic arteries in the interlobular areas, then in the central or hepatic vein zone, and finally in the peripheral or portal vein area. The infiltration continues its transformation process until the entire hepatic structure, including the connective tissue, is affected.

Etiology.—This disease is supposed to have a microbic origin, though a definite micro-organism has not yet been discovered. The staphylococcus and tubercle baccillus are claimed as possible causes. It is associated with prolonged suppuration, especially in connection with tuberculous affections of the bones and is an expression of a severe disturbance of metabolism rather than a primary disease. It is a feature of general amyloid infiltration, in which the spleen and kidneys are also implicated. Necrosis of the bone, hip-joint disease and pyelitis frequently precede the hepatic involvement. Pulmonary tuberculosis with cavity formation, tertiary syphilis, rickets, malaria, leukemia, pseudo-leukemia, malignant tumors, nephritis and infectious diseases are enumerated as probable causes.

Pathology.—The liver is uniformly and exceedingly *enlarged* and inspection may discover the unnatural prominence in the right hypochondriac and epigastric regions. Its *appearance* is waxy and the amyloid parts show a mahogany-red reaction when tested with weak iodin solutions. It is *smooth* and *hard,* the edges are rounded but not well defined, while the transverse fissure is exaggerated by the great enlargement. *On section* the oil droplets do not exude, though it may be associated at times with fatty infiltration, nodules may be discovered in certain syphilitic or cancerous forms and the amyloid change is sometimes circumscribed.

Symptoms.—The *waxy complexion* suggests the existence of some wasting disease. The *excessive enlargement of the liver* and the coexisting splenic increase, together with the amyloid kidney, which is determined by the albuminous urine and the waxy casts, will be the most confirming symptoms. In *children* maldevelopment and the prominent abdomen will be additional signs. *Gastro-intestinal disturbances* are often bothersome; jaundice is not present, but the stools are light colored because the diseased hepatic cells can not form bile. Pain is not a symptom unless it is associated with syphilitic hepatitis. A *sensation of weight* always exists on account of the liver's large size. Ascites is not always present unless there is a general hydremia. Mental disturbances are often marked.

Diagnosis.—The diagnosis is not difficult because the etiology is generally clearly established. Physical examination will disclose the pronounced hepatic enlargement and the increased area of hepatic dulness. The liver usually protrudes some distance below the free margins of the ribs. The absence of jaundice, the presence of dropsy, the enlarged spleen, and the amyloid kidney (discovered by the urinary examination) are positive confirming signs. The enlarged liver attending *leukemia* will be settled by the blood examination and the absence of the other symptoms of amyloid liver, while the organic growths will be differentiated by comparisons given in other chapters.

Treatment.—No treatment will apply when the disease has progressed to any considerable degree. The *primary cause* will require first attention, but that is generally known too late. The *diet* should be limited to nitrogenous foods and general measures should be instituted to keep the digestion in good condition. Stimulants must be prohibited. Alkalies have been lauded for their palliating influence, but they do not exercise any curative effect. *Ammonium chlorid* is most efficient in this respect. Nutrition and elimination must be watched carefully. When syphilis is a factor we may employ the *iodids, mercury, nitric acid* and *aurum chlorid.* In suppurating diseases we may gain much from *calcarea fluorica, iodoform* and *silicea.* The *tubercular causes* should direct our attention to the remedies and the treatment considered best for that disease. Symptomatic help is about all we may expect from remedies in a well advanced case.

PERIHEPATITIS.

Perihepatitis, otherwise defined as subphrenic pyopneumothorax, is an inflammation of the peritoneal covering of the liver. It is a secondary affection, being the result of inflammatory extension by continuity, a part of a generalized peritonitis, or a rare condition following a pleuritic extension through the diaphragm or a possible sequence from traumatism. It occurs as an acute suppuration or as a chronic inflammation of the perihepatic fibrous membrane with hyperplastic increase. The acute variety arises from the perforation of gastric or duodenal ulcers, hepatic diseases like abscess or hydatids, inflammations of the gall-bladder and from injuries to the liver. The chronic form is more liable to come from the pressure of tight lacing or injuries which develop subperitoneal adhesions, from cancer, gumma, hydatids or gall-bladder obstructions.

Pathology.—The peritoneal covering, particularly of the right lobe, is inflamed and thickened by a fibrous or puriform exudate with adhesions between the layer covering the liver and the diaphragm. The sac thus formed will contain a considerable quantity of pus, thus establishing a subphrenic abscess, or in case the diaphragm is perforated, forming a subphrenic pyopneumothorax. The pus in the abscess is mixed with bile and crystals of bilirubin and fatty acids. This is a valuable sign in the differentiation of subdiaphragmatic abscess and empyema. Rupture of the abscess may take place into the lung, stomach, intestine or peritoneal cavity. When the chronic form ensues, the thickening of the membrane is more pronounced and adhesions may form between the diaphragm, stomach, colon or abdominal wall and liver. The sclerotic contraction leads to hepatic atrophy and partial or complete occlusion of the vessels and the bile ducts.

Symptoms.—*Sudden and severe pain,* resembling peritonitis, characterizes the early symptomatology. It is localized in the right hypochondrium and epigastric regions. *Tenderness* is experienced on palpation and respiratory movements. *Chills* announce the abscess formation and a septic fever follows. *Gastric disturbances* are usually present and jaundice may be noticed should the bile duct suffer from compression. A *friction* rub of short duration may be detected in the mammillary line from the seventh rib downward and in the axillary line from the ninth rib downward. *Dulness* may extend as high as the angle of the scapula and the right hypochondrium may bulge while the lower intercostal spaces are motionless. The liver may be crowded downward from its normal position.

Diagnosis.—*Empyema* resembles acute perihepatitis, both by the physical signs and the symptoms. Empyema, however, is characterized by pleuritic pain associated with a cough and cardiac displacement to the left, while perihepatitis presents more abdominal symptoms. The liver is never crowded downward so much in empyema. Aspiration settles the differentiation, showing bile when the liver is involved. Pfühl states that in tapping a subphrenic abscess the spurting occurs with inspiration, while in empyema it takes place with expiration.

Prognosis.—The mild attacks terminate favorably and are of short duration. Much depends upon the character of the primary disease. The suppurative process may run a subacute course, causing a protracted illness and general exhaustion. The acute variety always presents the danger of rupture, while atrophy is the unfavorable feature of the chronic case. The signs of collapse are always unfavorable. Surgical interference accomplishes much by the evacuation of the pus.

Treatment.—The relief of pain must be induced by opiates and indicated remedies, while the inflammatory process may be localized by means of hot external applications. The febrile stage requires such remedies as *aconite, belladonna, bryonia* and *baptisia*. The suppurative process demands those remedies which are usually employed for that condition. A cure, however, can not be performed without the evacuation of the pus.

ABSCESS OF THE LIVER.

Suppurative hepatitis, or abscess of the liver, is a circumscribed collection of pus in the hepatic tissue. The majority of cases are traceable to an infectious cause which is conveyed through the portal circulation. A thrombotic infection enters a branch of the portal vein in the liver and gives rise to a suppurative pylephlebitis. A fragment of a thrombus may also enter the portal vein and act as an embolic cause of an abscess. The ameba coli, in the case of amebic dysentery, is sometimes conveyed from the intestinal tract into the liver and creates the necessary disorder for the development of suppuration. Mechanical causes, such as traumatism or obstruction by gall-stones, are considered as primary irritations, but, even in this case, the affection may be traced to a micro-organism.

An abscess may be established by an infectious embolus from the heart, lungs or systemic circulation which reaches the liver through the hepatic artery. Sloughing hemorrhoids are often causes of the infection ascending through the inferior mesenteric veins. Gastric ulcers, typhoid fever or appendicitis may create a portal pylephlebitis and thus develop an abscess in the liver. Foreign bodies, like parasites and round worms, may traverse the hepatic ducts and create a similar condition. In all probability, however, these agencies become associated with an acute pyogenic organism. Regurgitant embolism of the hepatic vein is also considered as a possible cause.

Pathology.—The liver is generally *enlarged,* the increased size encroaching upon the thoracic cavity. The right lobe is mostly affected near the convexity of the organ. *Prominences* may be felt on the hepatic surface if the abscess is near the costal border of the right lobe, or, in exceptional cases, in the left lobe. When the lower portion is involved the border of the organ becomes *irregular.* The *consistency of the tumor* is at first hard, but becomes softer as suppuration advances and a distinct fluctuation may be detected later. The *abscess formation* may be single and attain a large size, or it may appear in the multiple form as small abscesses. The amount of pus contained in a single large abscess may exceed a quart.

While the fluid represents a pus formation it may contain cellular debris, oil drops, leukocytes, cholesterin, fat and bilirubin crystals. On rare occasions the pus may be inspissated, caseous, calcified or encysted. Should *hydatids* be the exciting factor, echinococcus hooklets may be found. The amebae coli are often present. The *color of the fluid* varies from a grayish-white to a creamy or reddish-brown appearance. The *odor* is unpleasant, but depends entirely upon the infectious agency and the extent of necrosis. The *general appearance* of the liver gives expression to the circumscribed suppuration by the yellow areas beneath the capsule. When the disease depends on obstruction of the common duct by gall-stones, the gall-bladder and ducts will be greatly distended. The *hepatic cells* show the process of rapid degeneration by their altered shape and loss of nuclei. Round cell infiltration takes place about the blood-vessels which are filled with small emboli containing many staphylococci or streptococci, while liquefaction necrosis rapidly takes place. The *abscess may perforate* the diaphragm, involving the lung or pericardium, or, rupturing into the peritoneal cavity, set up a septic peritonitis; it may "point" on the abdominal wall.

Symptoms.—*Pain* is usually constant, and is located in the hepatic region, but may radiate to the right shoulder. In the *early stages* it is of a dull and boring character, but becomes more acute as the abscess develops. *Pressure* or a change of position generally aggravates it. The *temperature* is very high (103° to 105° F.) preceded by rigors and chills and followed by profuse sweats and exhaustion. It is liable to pursue an irregular and intermittent course. The *pulse* is rapid in proportion to the temperature. *Jaundice* is rarely observed, the skin being usually pale. When *perforation takes place* into the lung, a *cough,* with purulent expectoration, is an attendant symptom. *Loss of flesh* and gastro-intestinal disturbances are common symptoms. *Ascites* may develop when the inferior vena cava or portal vein is pressed upon. The spleen may be enlarged when the abscess occurs as the result of pylephlebitis or pyemic invasion. In *severe* and protracted cases delirium, subsultus, stupor and coma may intervene.

The *physical examination* reveals to the eye a marked prominence in the hepatic area when suppuration is fully established. *Palpation* detects the enlargement, tenderness and fluctuation. When the peritoneum is affected a friction fremitus may sometimes be felt. *Percussion* reveals enlargement upward.

Diagnosis.—The symptoms outlined above lead us to a correct diagnosis when they are confirmed by a thorough physical examination and the undeniable evidence of pus obtained by aspiration. Differential diagnosis must be a painstaking procedure. *Malarial fever* is differentiated by its history of malarial exposure, by its intermittency, by the quinin test, by the absence of a fluctuating tumor and by the cachexia. In this disease the spleen is always enlarged and the presence of the hematozoa of Laveran and free pigment is detected in the blood. Pus is not found in the liver. *Typhoid fever* is characterized by its peculiar temperature curve, the tympanites, delirium, rose spots and Widal reaction. The *echinococcus cyst* has less fever and requires a long time for its development. *Impacted biliary calculi* give the symptoms of biliary colic, jaundice and a history of stones in the feces may sometimes be obtained. *Carcinoma* and *hypertrophic cirrhosis* are differentiated in the chapters assigned to their consideration.

Pleural empyema causes the most doubt in diagnosis. While the physical signs are somewhat similar, a diminished fremitus and lessened vocal resonance, together with bronchial breathing from compression, will be invariably discovered. The contents of hepatic abscess show, in comparison, the microorganisms of suppuration, broken down liver cells, bile pigment and sometimes the ameba coli. The percussion flatness of empyema will also change when the sitting position is assumed. *Subphrenic abscess* is characterized by a marked downward displacement of the liver and the absence of hepatic debris, bile pigments, etc., on aspiration. *Abscess of the abdominal wall* is not affected by respiratory efforts as are hepatic enlargements. It is well to repeat that aspiration affords the most satisfactory differentiation.

Prognosis.—The result of this disease is generally regarded in an unfavorable light, and yet, except in multiple abscesses, surgical evacuation of the pus has reduced the mortality to a minimum. Unfavorable results may, however, follow this undertaking, but it is due to general sepsis already established.

Treatment.—Aside from the surgical procedure, the physician is called upon to relieve the results of general pyemic infection. In the beginning of the disease it is possible to give some aid toward localization of the pus by outward applications of heat, but the most efficient work of the physician, before and after evacuation by operation, is to contend with the debilitating effects of septic inflammation. *Stimulants* and nourishing food may be required for this purpose, and it is often necessary to give the heart more than usual attention.

Bryonia, baptisia, echinacea and *eupatorium* are efficient remedies in the early stages. When suppuration ensues we may rely upon *hepar sulphur, calcarea fluorica, sulphur, mercurius, silicea* and that class of remedies which have been heretofore mentioned for that purpose. *Strychnia, china, leptandrin,* and the *arsenate of quinin* are always to be considered.

ACUTE YELLOW ATROPHY OF THE LIVER.

Malignant jaundice, parenchymatous hepatitis and icterus gravus are the other terms by which this disease is known. It is characterized by extreme acute jaundice, profound toxemia and a rapid destruction of the liver tissue which terminates in fatty degeneration and marked atrophy.

Etiology.—The cause is evidently an infection or intoxication which is not, as yet, understood. *Pregnancy,* alcoholism and various micro-organisms have been claimed as the probable causes, but no definite information has as yet been obtained. While it may appear at any time of life, it seems prone to attack those of middle life most frequently. *Drugs,* like arsenic, phosphorus, mercury and antimony have been regarded as possible etiologic factors, but no satisfactory knowledge exists to substantiate the claim. *Malaria,* diphtheria, relapsing fevers, pyemia, syphilis and even powerful emotions have been ascribed as predisposing causes, but the fact exists that we are still in the dark regarding the origin of the disease.

Pathology.—The *liver cells* show evidence of rapid destruction similar to the condition in phosphorus poisoning. The *liver* is, therefore, very much reduced in size, though, in the beginning, it may be enlarged. The *capsule* is shrivelled and the tissue is of a pulpy consistency. The *color* is ochre-yellow, but presents areas with red and yellow discolorations. The *red appearance* represents an advanced stage and is due to an excess of blood in the capillaries and the pigment of destroyed red blood cells. *Microscopically,* we shall find, in addition to the necrotic process, that the nuclei of the cells have disappeared and the cell walls contain fat globules. Later the cells may show complete disintegration with the appearance of fat droplets, granular debris, cholesterin, leucin and tyrosin, together with crystals of bilirubin. The *heart* and *kidneys* may show similar degenerative changes, while the spleen may become greatly enlarged from active congestion. *Ecchymotic spots* may appear on the skin and mucous membranes, while *hemorrhage* from the stomach and intestine is not uncommon. *Dropsy* of the serous cavities, with edema of the brain and lungs, may be observed. The *blood* is diminished in quantity and somewhat dark in appearance.

Symptoms.—The *symptoms* are recognized only during the advanced stages of the disease. *Headache,* nausea and vomiting, with moderate fever, are the early premonitory symptoms, and in a few days jaundice appears, which does not differ materially from that of the catarrhal form. It first appears in the face and soon involves the whole body. In a few days, or possibly in a few weeks, serious nervous and cerebral symptoms are recognized. *Delirium,* which often becomes violent, restlessness, severe headache and convulsions are followed by stupor and coma. *Tremors* are pronounced and the jaundice constantly increases. The *liver* rapidly diminishes in size, while the spleen becomes enlarged. *Vomiting* is violent, epistaxis, hematemesis and hematuria are apparent, while the stools are devoid of bile. The *urine* is generally scanty, but very high colored; urea is decidedly diminished, bile pigment, albumin, tube casts, leucin and tyrosin are found by chemical and microscopic examination. Only a moderate rise of *temperature* is observed, but the *pulse* rapidly increases. Attending the rapid atrophy of the liver it is possible to determine a marked tenderness in the hepatic area.

Diagnosis.—The symptoms of the *second stage* of the disease, together with the sudden hepatic atrophy, make the diagnosis more certain. The increasing jaundice, the grave delirium and nervous excitement, the excessive bile elimination, the hemorrhages and particularly the presence of leucin and tyrosin in the urine are positive signs for diagnosis. *Acute phosphorus poisoning* offers the nearest comparison. In this, however, we usually discover the chemical cause, the hepatic atrophy is not so marked, the nervous symptoms are not so extreme and leucin and tyrosin are less commonly found in the urine. *Hypertrophic cirrhosis* has a more gradual onset, fever is rarely present, the liver slowly enlarges, while leucin and tyrosin are not found in the urine.

Prognosis.—A fatal result may be expected soon after the serious symptoms are discovered. Treatment does not offer any possible relief.

THE LIVER IN PHOSPHORUS POISONING.

Though we may not regard the results of phosphorus poisoning in the sense of a disease of the liver, nevertheless the pathologic conditions which ensue have such a close relation to fatty degeneration that it is well to give some attention to the study of the disease. It is not a common thing for a patient to take this poison, and yet this does occur. Those employed in match factories are particularly liable, and the extensive use of phosphorus in matches makes it a more common danger than would seem to be expected.

Symptoms.—*Nausea,* intense abdominal pain and vomiting of the gastric contents containing bile and traces of phosphorus are the common symptoms. *Intense jaundice* appears in a few days and the emesis may return with the characteristic black vomit. Serious *nervous symptoms* then develop in which convulsions, delirium and coma are recognized. The *urine* is scanty and contains bile, bile-acids, albumin, sarcolactic acid and, in exceptional cases, leucin and tyrosin. *Fever* is not present, while before death the temperature is liable to be subnormal.

Pathology.—The liver is enlarged at first, though atrophy may develop slowly; it is lighter in color, mottled in appearance, soft and friable in consistency. *Fatty degeneration* is in evidence when section is performed and the microscope shows disintegrated liver cells, fat globules, granular debris, bile coloring matter, cholesterin and sometimes leucin and tyrosin. Fatty and granular degeneration of the kidneys will be a coincident morbid condition.

Diagnosis.—It is often difficult to make a diagnosis of acute phosphorus poisoning from the symptoms alone. It is frequently confounded with *acute yellow atrophy,* but this latter disease has a different history, a slower onset and jaundice is an early symptom. The black vomit and the nervous symptoms also appear earlier and persist throughout the disease. Leucin and tyrosin are more commonly found in the urine than in phosphorus poisoning.

Prognosis.—The prognosis is invariably bad. Severe cases may terminate fatally in twenty-four hours. The average duration is from one to six days.

Treatment.—*Free emesis* is essential in the acute poisoning. *Sulphate* of *copper* (gr. 2-3) may be given every five or ten minutes, until ten grains are administered. It should then be prescribed in half-hour or hourly doses. If emesis is not obtained in this way, it may be necessary to use a powerful dose of *apomorphin muriate* (gr. 1/5) hypodermatically. When the degenerative process begins remedies are of no avail.

CANCER OF THE LIVER.

In the discussion of this subject reference is usually made to the development of carcinoma or sarcoma. Other morbid growths have been described, but the truly malignant enlargements are of this class. Angioma, adenoma, myoma and cysts are occasionally found. Syphilitic and parasitic invasions are discussed under separate chapters.

Etiology.—*Carcinoma* is a comparatively common disease of the liver. It is generally a secondary affection with the primary lesion, most frequently, in the stomach. It occurs generally during advanced life, though, in exceptional cases, it has been observed in children. *Males* furnish the greater proportion of cases, though it may appear in women as a secondary lesion following carcinoma of the uterus or breast. *Heredity* is a minor influence, though it is usually considered as an etiological possibility. *Traumatism* and mechanical obstruction should be regarded as occasional factors. The existence of primary carcinoma of the liver lacks authoritative confirmation.

Pathology.—*Cancer of the liver* will be recognized in the form of multiple nodules or as a general or massive infiltration. The former is the more common variety, the nodules varying in number, size and consistency. The majority of them are hard, while some are soft, the variation depending, to a great extent, upon the fibrous surroundings, the cell proliferation and the blood vessels which are concerned with the organic development. They are both primary and secondary, but they are larger and more numerous when secondary. In appearance they are usually opaque, white or yellowish white. *Vascular growths* have a red color. The superficial nodules project above the hepatic surface and may be felt through the abdominal wall in the emaciated subject. The center may undergo softening with a resultant cystic formation. *Fatty degeneration* in the center of the nodules may lead to absorption and contraction so that umbilication is present. The *capsule* covering the nodular growth also becomes indurated and may form adhesions to surrounding tissues.

The *massive form* is a primary infiltration of the liver and is found as a large tumor which affects a great proportion of the hepatic substance and, therefore, enlarges the organ decidedly. It is gray in color. The *radiating form* is made up of multiple small nodules but is not of frequent occurrence. A *colloid form* is a rare secondary development. *Cancer with cirrhosis* is another rare form. In this the liver is but slightly enlarged, is greenish-yellow in color and studded with small white nodules.

Degeneration of the cancerous nodules frequently occurs. It is essentially a fatty metamorphosis of the cells with occasional blood extravasations and a degree of suppuration. The *primary form* starts in the liver cells with the capillary network forming a stroma; an additional stroma is later added to this. The *secondary growths* are of embolic origin through the portal or hepatic veins. They start with a cancer cell of the capillary wall and gradually develop other cells throughout the hepatic parenchyma. The cells are mostly epithelioid in character, but may be polygonal or even cylindric. Various grades of fatty degeneration may be recognized. The trend of all carcinomatous growths is to enlarge the liver decidedly.

Sarcoma is invariably a secondary growth and includes melanosarcoma, lymphosarcoma and myxosarcoma. The first is the most common form; it is usually multiple, but may have a widespread infiltration throughout the liver and it may be associated with a similar melanotic distribution in different parts of the body.

Symptoms.—The *early manifestations* of hepatic cancer are not prominent in the average case. Much, of course, depends upon the location of the lesion and its relation to the primary cause. As it is so often associated with a primary growth in the pylorus, we should naturally expect the early appearance of gastric disturbances. A general depreciation of the health is also manifest and the cancerous cachexia is one of the guiding signs long before the tumor is discovered. In some cases, however, the *hepatic enlargement* with its nodular feel, is the earliest suggestion of the disease, and in other instances there are absolutely no reliable or distinctive symptoms until the disease is well advanced. The *patient* invariably complains of the loss of strength and flesh and there may be a sense of discomfort in the right hypochondriac and epigastric regions. The symptoms of the *primary disease* may at first overshadow those attending the carcinomatous development in the liver.

Jaundice is commonly present, though it may be absolutely wanting in many cases. As it is due to the compression of the smaller biliary passages, it is not intense. Bile, while not always absent from the feces, is fairly constant in the urine. The *presence of melanin* indicates the existence of the pigmented varieties of cancer. The accentuated features of jaundice appear when the larger ducts are obstructed. *Albuminuria* is uncommon. *Pain* is usually present and is dull and boring in character and generally located in the right hypochondrium. When there is associate biliary obstruction it may be exacerbating and extend toward the right shoulder. The *paroxysms* often increase as the hepatic enlargement develops and yet very large tumors may develop without marked pain. *Ascites* is not common; it takes place only when the portal vein or inferior vena cava is compressed or involved by cirrhosis with cancer, or when cancerous tissue develops in the peritoneum sufficient to cause effusion. When ascites is marked it often obscures the diagnosis of hepatic tumor. *Fever* is not generally observed until the later stages, and is irregular, intermitting and moderate. Blood examination shows hemoglobin reduction. Splenic enlargement is not often found. *Cerebral* symptoms such as headache, hebetude and delirium, sometimes characterize the advanced stages.

Physical Signs.—*Inspection* reveals an early enlargement of the superficial abdominal veins and later some prominence in the upper epigastric and hepatic area. The nodular growth may be visible in the advanced development when the patient is emaciated. *Palpation* detects the tumor when it extends below the line of the ribs. It is pushed downward by the diaphragm in forced inspiration. The nodules, and sometimes the central pits or depressions, are felt. The hepatic enlargement may also be estimated by palpation. *Percussion* outlines the liver enlargement and the various irregularities of the cancer, as they develop.

Diagnosis.—This requires not only a painstaking physical examination, but a careful inquiry into the individual and family history. The age and cachexia, the progressive enlargement of the liver, the localized pain, the characteristic nodules, the evidence of metastasis, the jaundice and the possible ascites will constitute the most satisfactory evidence of organic development.

Differential comparison must first be made regarding *cancer of the pylorus.* In the latter we shall more often find the tumor in the epigastric area, while the hepatic outline usually shows a distinct separation from the pylorus; this, however, is not easily determined when secondary hepatic involvement causes adhesions. Artificial dilation of the stomach will aid in the differentiation somewhat, and the examination of gastric contents will give some guidance; both of these, however, are often unreliable, but jaundice is generally absent. *Cancer of* *the gall-bladder* is recognized as an adnexum of the liver by palpation which defines its shape and superficiality, and also by percussion when the stomach is artificially distended with gas. The signs of obstruction may also settle the location of the tumor. *Gall-stone impaction* and also catarrhal affections of the gall-bladder are to be differentiated by the more acute development of symptoms, the gastric disturbances, the absence of hepatic enlargement and the absence of cachexia, ascites and general dropsy. *Intestinal tumors* in the ascending colon or hepatic flexure are distinguished by the doughy feel and the relief by purges. In case of fecal impaction, the accumulation of gas above the tumor, the occasional change to a tympanitic note and the abdominal distention will differentiate. They are not affected by the descent of the diaphragm during respiration and the pain is more acute. Tumors of the intestine, particularly of the transverse colon, are more movable.

Tumor of the right kidney may present a perplexing similarity of symptoms. This may be settled by bimanual palpation. The ascending colon, which covers the kidney, will, however, take an oblique course, from above downward, and when empty it gives a tympanitic note. The bulging of the ribs is not so pronounced or uniform as in hepatic enlargement. Examination of the urine may also, at times, furnish deciding evidence. The *fatty liver* is softer than the cancerous and presents its other characteristic features. *Amyloid liver* is smoother, and the rounded border is often felt. It is associated with a similar enlargement of the spleen. *Hepatic abscess* gives a different history; it occurs at any age; hectic temperature, chills and sweats are distinct features, pain is sharper, and anemia takes the place of cachexia. A fluctuating tumor is sometimes discovered, the enlargement is upward, the duration is shorter and aspiration may give an opportunity for decisive bacteriologic proof. *Echinococcus cysts* have softer nodules, the emaciation is less and the spleen is enlarged and the characteristic hooklets are found by aspiration. It is, moreover, a rare disease. *Hypertrophic cirrhosis* occurs in younger people, has no cachexia, gives little hepatic tenderness, has no nodular enlargement and an alcoholic history is usually obtained.

Benign growths like adenomas, angiomas and cystomas are also to be considered in the study of hepatic tumors inasmuch as they are so often mistaken for carcinoma of the liver. They may occur, however, at an earlier age, no primary growth is discovered in other organs, the cancerous cachexia and wasting are not in evidence and the other characteristics of malignant disease are absent. *Sarcoma* does not present many differentiating symptoms so far as the hepatic tumor is concerned. The primary involvement elsewhere and the peculiar morbid phases which typify the various forms of this organic increase will be recognized in its pathologic makeup, and this is all that is necessary for a general diagnosis. Evidence that a cancer is secondary will be established when signs of its invasion are discovered elsewhere.

Prognosis.—The disease is always fatal and the prolongation of the suffering is dependent upon the patient's endurance and the progress of the pathologic changes. *Treatment* is only palliative.

CIRRHOSIS OF THE LIVER.

Cirrhosis of the liver is a chronic hepatic disease which is characterized by an excessive formation of connective tissue, destruction of the parenchyma, a harder consistence, together with atrophy or hypertrophy of that viscus. In some cases there will be no particular decrease or increase in size. It has been variously termed sclerosis of the liver, gin-drinkers' liver, interstitial hepatitis and granular liver.

Pathology.—Three pathologic varieties are presented: (a) atrophic cirrhosis, (b) hypertrophic cirrhosis and (c) biliary cirrhosis.

Atrophic Cirrhosis.—This is the most common form and its cause is supposed to be traced to alcoholic excess. In the early stages the liver is enlarged. Later it gradually becomes atrophic, hard, granular and changed in contour. Section shows the developing bands of connective tissue which, surrounding different areas of the parenchyma, may compress and crowd these isolated portions to the surface, giving rise to the term "hob-nail liver." The *connective tissue formation* begins about the terminal branches of the portal vein which, in conjunction with the liver cells, are compressed by the constant increase of tissue. The lobular circulation is, therefore, gradually cut off as the connective tissue proliferation eventually produces contraction. The *hepatic cells* suffer therefore from compression, which leads to atrophy; they may also undergo fatty metamorphosis, on account of which the hepatic tissue softens and assumes a yellow appearance. In some instances the liver becomes smooth from a more uniform distribution of connective tissue throughout the parenchyma. *Fatty infiltration* may occasionally overbalance the contraction sufficiently to enlarge the liver. Amyloid degeneration sometimes characterizes the infiltration.

Hypertrophic Cirrhosis.—This is often termed elephantiasis of the liver, inasmuch as the organ increases decidedly in size. It is often confused with the first stage of the atrophic variety. The overgrowth of connective tissue shows no tendency to contract and, therefore, the branches of the portal vein are not compressed, but the hyperplasia invades the biliary tract causing obstruction. Jaundice is, therefore, a prominent symptom. Proliferation of bile-ducts takes place. The *liver,* instead of being reduced in weight, as in the atrophic form, from four or five pounds to two or three pounds, is actually increased to eight or ten pounds. It is still claimed by competent observers that this variety eventually contracts and assumes the atrophic form.

Biliary Cirrhosis.—The French writers have described this form of cirrhosis as opposed to the portal cirrhosis or atrophic variety. It follows from the obstruction of bile ducts, the retained bile, acting as a chemical irritant, starts a cirrhotic process around the small bile ducts. Pigment granules are deposited in the interlobular connective tissue and also within the acini. The liver structure becomes harder and the organ is very much enlarged.

Etiology.—*Atrophic Cirrhosis.*—Alcohol must still be regarded as the prominent cause of this form of cirrhosis, notwithstanding the fact that many cases are found among those who never indulged in spirituous drinks. While micro-organisms are supposed to cause it, no definite discovery has been announced in this respect. It is, no doubt, associated with infectious diseases. Certain *chronic diseases,* like syphilis, diabetes, gout, malaria, carcinoma and tuberculosis play an important part in the hepatic connective tissue increase. *Cardiac lesions* and obstructive lung diseases are factors to be considered at all times.

Hypertrophic Cirrhosis.—The causes which lead up to this form of cirrhosis are not understood so well as those of the atrophic variety. The male sex is mostly predisposed, though alcoholism probably has little influence. It has been observed in young adults following infectious diseases. Catarrhal jaundice is, no doubt, a factor. Hypertrophic cirrhosis is discovered most frequently among inhabitants of the warmer climates. *Biliary cirrhosis* follows chronic obstruction of the bile ducts.

Symptoms.—*Atrophic Cirrhosis.*—The positive symptoms of atrophic cirrhosis do not appear early. When they do appear they are, in reality, the secondary expressions of the pathologic condition. Thus *anorexia,* nausea, a sense of distention and discomfort, constipation, slight jaundice and a gradual loss of flesh are due to the slowly developing atrophic changes which are thus manifested first in the disturbances of the alimentary canal. As the gastro-intestinal mucosa suffers a more intense congestion these symptoms will be exaggerated to the extent of increased nausea and vomiting and the possible occurrence of hemorrhages from the nose, stomach and intestine. The *tongue* is invariably covered with a foul and thick coating. *Hemorrhoids* resulting from portal obstruction, uterine hemorrhage and hematuria are also frequent occurrences, but above all we shall notice *marked abdominal dropsy.* This may assume large proportions, causing a decided abdominal protrusion. *Emaciation* increases as the disease progresses, the facial expression is pinched, the superficial veins are prominent, the nose is often red and the skin is of a sallow color. The *spleen* becomes enlarged and, as the result of toxemia, there may be *nervous symptoms* such as delirium, convulsions, stupor and coma. *Fever* is not a common symptom. The *urine* is scanty, has a high specific gravity and contains bile and urates, while albumin and casts may be present. The urea is always low.

Physical examination reveals on *inspection* abdominal distention, enlarged abdominal veins and a sallow skin. *Palpation* is not satisfactory unless the fluid is removed by tapping. In this case we shall find the liver in advanced stages much smaller, while the spleen is correspondingly enlarged. Sometimes on deep palpation the under surface may be felt. *Percussion* discloses the diminished area of hepatic dulness and the corresponding increase of splenic dulness. Due allowance must be made for the early enlargement and the similar permanent condition in fatty cirrhosis.

Hypertrophic Cirrhosis.—The alcoholic history is not discovered and the disease may appear in younger people. *Jaundice* is an early symptom and increases as the disease progresses. *Fever* is sometimes an attending symptom and paroxysms of pain often simulate biliary colic. Inasmuch as the portal circulation is not disturbed so much as in the atrophic form, the hyperemic conditions of the gastro-intestinal tract are not so marked and, hence, gastric and intestinal disturbances are less prominent. *Hemorrhoids* are not so constant, the *spleen* is but slightly enlarged and ascites does not usually appear. *Bile-stained urine* is more common than in the atrophic variety, but blood is not generally present. The urea is normal in amount and albumin and tube casts are discovered only in advanced cases. The *feces* show the presence of bile at times and again it may be entirely absent. The *red blood corpuscles* are decidedly diminished in number and there is a relative leukocytosis. The disease usually runs a rapid course. Severe symptoms like icterus gravis, high fever, hemorrhages, delirium and coma may precede death.

Physical examination reveals a decided, uniform and smooth enlargement of the liver, the lower border of which may be easily felt below the free margin of the ribs. The increased size may be confirmed by percussion.

Biliary Cirrhosis.—The signs of this form of cirrhosis are closely related to the cause which is some form of obstruction of the bile ducts. This may arise from the impaction of gall-stones, a tumor or a stricture of the duct. Following this the features of catarrhal or suppurative cholangitis are liable to appear. *Jaundice* is even more pronounced than in the hypertrophic form and is one of the earliest symptoms. *Intermittent fever* is another symptom.

Diagnosis.—*Atrophic Cirrhosis.*—The atrophy of the liver, the ascites and the absence of acute symptoms, together with the confirmation of the symptoms previously outlined, should be sufficient for diagnosis. *Tuberculous peritonitis,* with its marked effusion, is often confused with this disease; the abdominal tenderness, the fever and evidences of tuberculous involvement elsewhere aid in the differentiation. The peritoneal effusion in cirrhosis is usually greater than that of peritonitis.

Hypertrophic Cirrhosis.—This must be compared with the diseases in which the liver becomes enlarged. In *cancer* the patient is usually over forty, the enlargement is uneven, ascites is more common, the spleen is not enlarged and alcoholism has even less influence. In *amyloid liver* there is a similar splenic enlargement, but there is neither pain nor jaundice and the etiology of amyloid disease is quite clear. *Echinococcus cyst,* though presenting many similar symptoms, is defined by its history, which points to a definite infection, the absence of marked emaciation and leukocytosis, and the physical finding of the irregular hepatic surface with the hydatid thrill. *Chronic biliary obstruction* gives a history of biliary colic, early jaundice and other evidences of biliary obstruction. It runs a more rapid course and is liable, in chronic cases, to terminate in atrophy. *Hepatic abscess* has a traumatic or pyemic history, localized pain is severe and constant, tenderness is marked, hectic fever with chills and sweating are early features, the course is acute, the tumor is fluctuating and pus is obtained by aspiration.

Prognosis.—Hepatic cirrhosis is not a curable disease. Improvement depends upon the extent of the cirrhosis and the removal of the cause. If uninterrupted, it pursues a progressive course until improvement is beyond hope. The disease may remain in comparative abeyance when the irritating cause is eradicated and the compensating circulation is thoroughly established. Death will take place in a few months or at least a year after the pathology is completely established. Ascites will be the most troublesome symptom.

Treatment.—The *alcoholic habit* should be stopped and a reasonable amount of exercise should be instituted. The *diet* must be of the simplest form and in many instances it will be best to use milk, Koumiss and light nitrogenous foods; the starches and sugars should be restricted. *Alkaline mineral waters* and possibly purges will be beneficial in many respects. The latter should be employed with extreme caution, as hepatic excitement is often created when these measures are too vigorous. *Cascara, iridin* (gr. 2-5) or *chinoidin* (gr. 4-6), are generally sufficient for this purpose. It is possible that moderate doses of *calomel,* frequently repeated, followed by a saline, will give at least temporary improvement in many cases. *Elaterin* (gr. 1/20-1/10) may be used three or four times daily for the purpose of obtaining profuse watery stools. By the persistent use of aperient waters it is possible to relieve the portal congestion for a short time at least. Diuretics like *apocynum, digitalis, potassium acetate, convallaria, hydragogin* and *diuretin* may be of service, particularly when the cardiac action is weak. In such cases I believe the employment of *strychnia* (gr. 1/60-1/40) four times daily is quite essential.

The remedies pertaining to the gastric and intestinal disturbances have already been considered under separate topics. In this respect we shall often find use for *arsenicum, nux vomica, bryonia, cinchona, magnesia, podophyllum, iris* and other remedies. Gastric lavage may be indicated as in catarrh of the stomach. Remedies, which refer to the tissue changes in the liver, like *phosphorus, mercurius, chelidonium* and the *iodids* may be employed when the symptomatology calls for them. Goodno is partial to the use of *aurum muriaticum* in the first or third decimal triturations and to this we might also add *argentum nitricum, nitric acid, picric acid* and *plumbum.* Gaillard reports a case of cirrhosis of the liver which received no benefit from internal medication, but which was much improved and finally cured by the ingestion daily of 150 gm. (5 oz.) of pig's liver.

Unfortunately we rarely obtain much benefit by the use of our remedies and, in the advanced cases, we can not expect to relieve the ascites by any such measures. This will invariably require the operation of paracentesis abdominis, which is essentially a surgical procedure and demands strict aseptic precautions as well as surgical judgment. Care should be exercised in order to avoid cardiac failure and vasomotor paralysis which are always possible dangers; for this reason the ascitic fluid should not be withdrawn too rapidly. To perform this it will be best to allow the patient to sit up or assume a semi-recumbent position when the trochar is used. The after-treatment of the wound should be in accordance with accepted surgical principles. The removal of the fluid gives at least temporary relief, but in no sense cures the disease. The secretion is rapidly renewed and it has been my observation that as soon as tapping is instituted the debility of the patient increases and the peritoneum fills more rapidly.

DISEASES OF THE PANCREAS.

PANCREATIC HEMORRHAGE.

Pancreatic apoplexy has been considered as a distinct entity only during the past few years. Aside from its relation to acute pancreatitis, it must be recognized as a cause of death in a small proportion of cases. When there is extensive hemorrhage the entire glandular tissue may be destroyed, while the blood may also invade the retroperitoneal space or, if the peritoneal covering is broken, it may enter the lesser peritoneal cavity. Slight hemorrhages which are due to chronic passive congestion, hemophilia, purpura or acute infections are not included under the head of pancreatic apoplexy.

The causes of these hemorrhages have not yet been ascertained though, no doubt, a necrotic process must be fundamental. Traumatism has been claimed as a factor and the atheromatous state, encouraged by alcoholic excesses and a rich diet, must be taken into consideration. The corrosive action of an excessive or perverted pancreatic secretion has been claimed by some as a cause. Acute pancreatitis and carcinoma must always be counted upon as possible primary causes. The majority of cases occur in patients of advanced years.

Symptoms.—The symptoms, as described by Prince, appear suddenly and without warning. The patient complains of severe pain with a sense of constriction in the epigastric region. Nausea and obstinate vomiting immediately follow, but no relief is experienced. Tympanites is often an early symptom and tenderness may be detected, particularly in the epigastrium. The patient becomes anxious, restless and depressed, while a cold sweat, a small, weak and rapid pulse, with anemia if the hemorrhage is large, will confirm the suspicion of sudden internal hemorrhage. With profound prostration and syncope death may suddenly take place. The suddenness of this is said to be due to shock through the solar plexus.

ACUTE PANCREATITIS.

This disease is exceedingly rare. We are indebted mostly to Fitz for our knowledge regarding it. He recognized three varieties, the hemorrhagic, the suppurative and the gangrenous, but as these represent different stages of the same disease the study of each may be undertaken under this one topic. The fibrous and fatty interstitial tissue is affected by the inflammatory invasion.

Etiology.—Definite conclusions respecting the etiology can not be given inasmuch as it is so rare that extensive study has not been given to the subject. Hemorrhage into the pancreas may result in localized inflammation, while traumatism, glycosuria, alcoholism and gastro-duodenal catarrh have each been deemed a factor. It is claimed by Opie that it may be due to bile entering the pancreatic duct during an attack of biliary colic, while there is no doubt that inflammation may extend from the duodenum to the pancreas. It has been found at autopsy in cases of acute tuberculosis, the specific fevers and pyemic conditions and, therefore, it has been ascribed to an infective agent. Males of advanced age seem to be mostly predisposed to this disease.

Pathology.—In the early or hemorrhagic stage the pancreas is infiltrated with blood either in the head or throughout the body, giving a red color to the organ. In case of small hemorrhagic foci we may find alternating spots of fat necrosis and blood accumulations. The hemorrhage may also extend into contiguous tissues and the mesentery, mesocolon, omentum and more external parts may be involved. Round cells and blood cells are found in the ducts or acini. Coagulation necrosis may be observed in some of the lobules and bacteria are usually abundant. In a few days the suppurative or gangrenous process supervenes. When suppuration occurs the pancreas generally becomes enlarged, very hyperemic and studded with small abscesses. The suppuration may be diffuse or limited to one large abscess should the inflammation tend to become chronic. Surrounding tissues may be invaded, the liver may become involved and thrombosis of the splenic and portal veins may ensue. When the gangrenous process develops we shall find a part or the whole of the organ degenerated into a dark slate-colored mass which is very soft and shreddy. This degeneration may begin as soon as the hemorrhage occurs and in a comparatively short time the organ may suffer complete metamorphosis and the dark and purulent extravasations be discovered in neighboring structures. Fat necrosis, peritonitis, peritoneal adhesions and thrombosis of the splenic vein may be accompanying conditions.

Symptoms.—Acute pancreatitis begins suddenly and generally without premonitory symptoms or evidence of an exciting cause. Severe *abdominal pain* is the first suggestion of its presence and this is located in the epigastrium but may extend into the upper umbilical area. *Nausea* and persistent *vomiting* are early symptoms, and the ejected matter is either bilious or black. *Constipation* is often obstinate, though diarrhea may be present. *Tenderness* may be elicited over the pancreas while tympanites is usually present. The *temperature* is not high and in some instances it may be subnormal. Death usually takes place in two or three days, or at most within a week with the appearance of sudden collapse.

In the suppurative form life may be prolonged for several weeks, and the symptoms will be similar, with the exception of collapse. In a few days, however, there will be an elevation of temperature which will be characterized by its irregular type and attended with rigors. The *temperature* may reach 104° or 105° F. *Slight jaundice* may be observed and hepatic and splenic enlargement are not uncommon. *Emaciation, increasing debility,* diarrhea, ascites and anasarca are progressive symptoms. The usual exacerbations and remissions of septic fever are liable to appear and a deepseated peritonitis may exist with its greatest intensity in the epigastric region. In the gangrenous form the gland may become entirely destroyed, the slough remaining in the peritoneal cavity or be discharged through the bowel. The symptoms do not differ from those of the suppurative form. Progressive weakness, localized peritonitis, with vomiting and diarrhea, will eventually terminate in death, which may be preceded by sudden collapse.

Chronic Pancreatitis.—This may result from some antecedent inflammation of that organ, but is more frequently due to catarrh involving the pancreatic duct. Diseases of the biliary passages may produce it by contiguity, while alcoholism, syphilis, tuberculosis and diabetes have been credited as causes. The pathology consists of an interstitial fibrous overgrowth resulting in permanent sclerosis and atrophy. It may show an irregular distribution with localized areas of sclerosis making the surface of that organ uneven. The ducts are dilated, tortuous or generally compressed.

Symptoms.—These are not prominent and point more particularly to the causative disease. The signs of pancreatic involvement is manifested only by slight paroxysms of deep epigastric *pain.* The patient may have an expression of anxiety and at times complain of *faintness. Ascites* and *jaundice* are occasional symptoms when compression affects the portal circulation. Free fat in the bowel movements and glycosuria are said to be present in some cases. Circumscribed resistance in the pancreatic area may be determined in some cases. Progressive debility and exhaustion are eventually noticed.

Diagnosis.—The diagnosis is rarely made before death. The appearance of the foregoing symptoms, and particularly circumscribed tympany, will be guiding points in making a diagnosis. *Obstruction* of the small intestine rarely takes place in this region, fecal vomiting would develop, and tympany would be more general. Obstruction of the large intestine may be settled by palpation, injection and inflation. *Perforation of the stomach* will give symptoms of gastric ulcer, while *biliary colic* may be differentiated by the more intermitting pain and the other distinctive symptoms of biliary obstruction. *Corrosive poisons* may be excluded by the history and examination of the mouth and the vomitus. *Acute gastro-duodenitis* will present the history of some stomach disturbance, a gradually developing fever, with the absence of the prostration and sudden collapse in pancreatic inflammation.

Prognosis and Treatment.—The disease is exceedingly fatal and its duration is short. A cure is an apparent impossibility. Surgery is a last resort, and some favorable records are given in this respect. Drainage may possibly save life when there are no inflammatory degenerations in the pancreatic structure. Medicinal treatment is not indicated.

CARCINOMA OF THE PANCREAS.

This disease is not common and primary cancer of the scirrhus variety is most frequently discovered. Colloid degeneration is a rare occurrence. It sometimes exists in the secondary form as an extension from the stomach, or duodenum. The head of the pancreas is usually affected and the growth may attain a large size. Metastatic involvement may cause an invasion of the liver, gall-bladder, lymph glands, peritoneum, stomach and the portal vessels. It occurs most frequently among men of advanced years, though it has been observed in childhood.

Symptoms.—Reliable symptoms are rarely obtained, particularly in the earlier stages. *Progressive emaciation* will be in evidence as the disease advances, and attending this there may be epigastric pain or digestive disturbance. *Cachexia* and the gradual loss of strength will be observed. *Vomiting* and *diarrhea* may become uncontrollable and collapse may suddenly appear. *Jaundice* is a frequent symptom when the enlarged pancreatic head obstructs the common bile duct, "and may be associated with an enlargement of the gall-bladder and liver. *Ascites* may develop when the portal vein is compressed and chylous ascites has occurred when the thoracic duct is compressed by the tumor. When the pressure affects the vena cava, dropsy may appear in the lower part of the body; *edema* of the lower extremities may also be present; should the duodenum be enlarged, gastrectasis may be a symptom; in other instances the compression may cause symptoms of intestinal obstruction. *Hydronephrosis* of the left kidney may be a result of pressure on the left ureter in case the pancreatic tail is the seat of the organic growth.

When emaciation is well advanced it is usually possible to detect a fixed tumor in the pancreatic region. If this is associated with fatty or greasy stools and the symptoms above are in evidence, we may diagnose the presence of pancreatic cancer. It is not, however, an easy matter in the average patient to define the tumor by palpation. Glycosuria is regarded as an occasional sequela and indican may be present in large amount in the urine. The presence of undigested meat fibers in the stools is conclusive evidence of impaired pancreatic digestion.

Diagnosis.—In making a diagnosis we must eliminate the following diseases: *Abdominal aortic aneurysm* will be simulated by the aortic pulsation caused by the pancreatic enlargement. In aneurysm, however, the impulse is expansile, while the cachexia, debility and hardness of pancreatic cancer will establish the distinction. *Carcinoma of the pylorus* offers the following distinctive history: The tumor is more movable, while dilatation and other gastric symptoms are recognized. The coffee-ground vomitus, the absence of HCl and the presence of lactic acid in the stomach contents, the black stools following hemorrhage, the absence of jaundice and ascites, and the location of the tumor in the pyloric area are sufficiently differentiating symptoms. The chronic course of pyloric growths should always be compared with the rapid development of pancreatic cancer. *Tumors of the transverse colon* are more superficial and movable. Inflation will usually locate the tumor and there will be other substantiating signs of intestinal obstruction. Sarcoma, adenoma, lymphoma and tuberculous growths may also invade the pancreas.

PANCREATIC CYSTS.

Pancreatic cysts are caused by an obstruction of the main duct or its branches. Calculi may, by their impaction, establish small cysts; cicatricial contraction of the pancreatic structure may cause stenosis of the ducts, while the closure of Wirsung's duct by concretions or inflammatory contractions may cause retention cysts of various sizes. Tumors of adjacent viscera and biliary impaction play a minor part in their presence, and traumatism, no doubt, has a direct or indirect influence upon their development. Men and women are affected alike, while adults furnish the larger number of cases. Congenital cysts have been reported.

Pathology.—The cysts may be single or multiple, varying decidedly in size. In some cases a single cyst may contain several gallons of fluid. The walls are generally thin, though in long standing cases the connective tissue formation may decidedly thicken and indurate the enveloping structure. The inner surface of the wall is smooth, trabeculated or roughened by thickened patches. Secondary cysts often communicate with the original one. The cavity contains a fluid which is viscid, alkaline and of high specific gravity (1010-1024). In the early stages the contents resemble the normal pancreatic fluid. The hemorrhagic feature is usually recognized and in some instances a hematoma may be converted into a serous cyst. Analysis of the cystic contents, obtained by exploratory puncture, show it to consist of epithelium, blood corpuscles and the ferments which transform starch into sugar and emulsify fats. The fluid is albuminous in character.

The cysts may develop rapidly or, in exceptional cases, pursue a very chronic course. It is possible for them to become stationary. The enlargement of the cysts may produce atrophy of surrounding tissues, crowd the stomach out of place or form extensive adhesions to its posterior wall. The contents of a large cyst may rupture into the stomach or the peritoneal cavity.

Symptoms.—The diagnostic symptoms are rarely discovered until the *tumor* has attained considerable size. *Pain* may then be experienced in the epigastrium, the left hypochondrium or about the left shoulder. When the tumor is large *jaundice* and *ascites* are not uncommon. *Nausea, vomiting, constipation* or *fatty diarrhea* are occasional symptoms. The stools are clay-colored and offensive while albumin and sugar may be found in the urine. As the cyst attains a large size, *local tenderness* or a sense of pressure is experienced in the epigastrium. Prostration typifies the general condition, and emaciation becomes noticeable.

Physical examination reveals a smooth and elastic tumor to the right of the median line when the head is involved, and to the left of the same point when the growth is in the tail of the pancreas. When the cyst is very large it will cover a larger area and fluctuation may be elicited. It may be slightly movable during inspiration and it is often possible to grasp it in the hand. A murmur may be detected by auscultation when there is aortic compression. The results of mechanical pressure depend upon the size of the cyst.

Diagnosis.—The physical examination, the deep-seated tumor behind the stomach, the character of the pancreatic fluid obtained by aspiration and the fact that it will digest albumins and emulsify fats will substantiate the diagnosis. *Malignant tumors of the pancreas* have early emaciation, cachexia and pain; the tumor is solid and has an irregular surface. *Aneurysm* will be distinguished by its expansile pulsation and the fact that pulsation in the case of pancreatic cyst is relieved when the patient assumes the genu-pectoral position. *Hydro-* or *pyo-nephrosis* will be determined by the history, the urinary examination and the lower and more lateral location of the tumor. *Echinococcus cysts* are usually multilocular while the pancreatic cyst may be either multilocular or unilocular. The former is also defined by the peculiar fremitus and the presence of hooklets in the aspirated fluid. *Circumscribed peritonitis* gives a history of sudden onset, fever, pain and localized tenderness, while *ascites, dropsy of the gall-bladder* and *ovarian cyst* must be determined by their characteristic differentiations.

Prognosis and Treatment.—The danger of rupture, hemorrhage and suppurative inflammation are always present. Under surgical treatment, which is our only resource, a fair number of cases may recover after proper drainage.

PANCREATIC CALCULI.

Pancreatic calculi are due to the precipitation of the inspissated pancreatic juice, the cause of which is unknown. A catarrhal invasion of the duct is without doubt predisposing, while pancreatic cysts and other forms of obstruction have, no doubt, an influence. It is a very uncommon condition. The concretions have the chemical composition of calcium carbonate or phosphate, they are grayish-white in color and they may be very small or relatively very large in size, and irregular in shape. They are liable to create dilatation of the ducts, abscess formation, chronic pancreatitis and fistulous openings into surrounding parts. The secondary changes in the pancreas may terminate in atrophy.

Symptoms.—Paroxysms of pain, similar to gall-stone colic, will develop when the calculi are passed from the duct into the duodenum. The pain generally radiates to the left side and is deepseated. *Free fat* is found in the stools and *glycosuria* is invariably present. The calculi may be found in the stools. Jaundice is a rare symptom and when present the differentiation from gall-stones is somewhat difficult. A conservative prognosis must be given and the treatment will be symptomatic and palliative. Operation should be considered.

DISEASES OF THE SPLEEN.

These are for the most part secondary to other diseases, particularly blood diseases, and their study will naturally come under the head of other affections. There are, however, a few conditions, distinctively splenic, which require separate discussion.

Dislocation of the spleen or floating spleen may be the result of abnormal enlargement, tight lacing, traumatism or relaxation of the splenic ligaments. Abdominal ptosis and pressure from the enlargement of the left lobe of the liver may drag or force the organ out of place. The symptoms may suggest inflammatory conditions of neighboring organs, but usually are indicative of pressure. Physical examination will detect the pendant organ below its normal location. *Percussion* reveals the absence of the normal area of dulness, while *palpation* locates the mobile tumor and detects its peculiar outline.

The *diagnosis* must differentiate the floating spleen from splenic enlargement, and a movable kidney must be excluded by its exclusive physical signs. In case of abdominal tumors the normal splenic dulness is unchanged. The prognosis is not generally unfavorable unless twisting of its vessels leads to necrosis, or intestinal obstruction follows from pressure. The treatment consists of mechanical means of support or possible resort to splenectomy.

Splenic Hyperemia.—Acute hyperemia exists as the result of the acute infectious diseases, amenorrhea, or in many cases from injury. The spleen rapidly enlarges except in cases of circumscribed hyperemia. It is darker in color and softer in consistency than normal.

Chronic or Passive Hyperemia.—This follows any obstruction of the portal circulation and may, therefore, be due to pressure from tumors, or to enlargement of the heart or liver; pulmonary disease and pylephlebitis may also be contributing factors. The organ is enlarged, firm and dark red in color; the capsule is somewhat thickened.

The *symptoms* are expressed by a sense of weight, fulness and pressure. *Tenderness* may be discovered by palpation and the spleen may be found below the free margin of the ribs; *percussion dulness* may be increased in area over the splenic region. The extent and character of the symptoms depends upon the causative disease.

New Growths in the Spleen.—Gummata, carcinoma, sarcoma, hydatids and tubercles are associated with similar formations in other parts of the body. They are of exceedingly rare occurrence and are usually discovered only at autopsy.

Amyloid Degeneration of the Spleen.—This is generally secondary to a similar degeneration in the liver and kidneys. It may attend such wasting diseases as tuberculosis, empyema, hip-joint disease, osteomyelitis or syphilis. The spleen is enlarged, smooth and yielding to the touch. In the "sago" form of degeneration the Malpighian bodies on section resemble sago granules; in the *diffuse* form the whole parenchyma presents a waxy appearance. The symptoms are defined by the general waxy appearance, the enlargement of the organ and the size of amyloid degeneration in other organs. The prognosis is unfavorable and the treatment conforms to that in similar involvements of other organs.

Hemorrhagic Infarct of the Spleen.—This may appear in the non-infectious form following an embolism from the heart in case of endocardial involvement or from clots from large aneurysms. Infectious hemorrhagic infarct usually creates an abscess. In the latter case chills, fever, vomiting and painful enlargement are present.

Splenitis.—This is an inflammation of the spleen which may be acute or chronic, and may assume a hyperplastic or suppurative character. The *acute* form develops after acute infections like typhoid and malaria. The *chronic* form follows repeated attacks of splenic anemia, passive congestions and leukocythemia. The *suppurative* form is due to infectious hemorrhagic emboli as described under hemorrhagic infarct.

The *symptoms* are similar to those of other splenic disorders, though the sense of weight, tenderness and tension may be magnified. Septic fever is present in the infectious form. Sudden pain will be experienced in the gastric region and it may be followed by the vomiting of pus and blood when an abscess ruptures into the stomach. Enlargement, increased dulness on percussion, and fluctuation may be detected by physical examination.

Diagnosis and Prognosis.—If the findings of the primary disease are carefully studied, the secondary splenic enlargement will not be mistaken. In case of abscess formation an exploratory puncture may settle the diagnosis. The enlargement of *chronic splenitis* often resembles the growths which affect neighboring organs and this will call for a careful discrimination of symptoms and physical signs. *Pleural effusions* of the left side and *fecal impaction* of the splenic flexure of the colon must be differentiated by the signs already discussed under those subjects. The results of splenitis depend decidedly upon the primary cause. An abscess is liable to rupture and cause general peritonitis. The chronic non-infectious form may pursue a harmless and protracted course.

The *treatment* also pertains to the primary cause. *Infections* must be treated in accordance with the principles already outlined in that respect. *Abscess* will require surgical interference; and splenectomy may be advisable in rare instances, though the results are not always encouraging. Much depends upon the physical conditions and operation is never to be undertaken in leukemic enlargement.

DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Etiology.—Acute inflammation of the peritoneum may be primary or secondary in character, may be circumscribed or diffuse, simple or infective. Pathologically it is characterized by an inflammatory exudate. The average case is recognized as a secondary peritonitis, having its origin in contiguous organs, or resulting from traumatism. The latter, however, has been reduced to the minimum on account of aseptic surgery in abdominal operations and the care of wounds. Peritonitis is not uncommonly the result of perforation in gastric ulcer or cancer and it may be a sequence of intestinal perforation following typhoid fever, appendicitis and dysentery. Diseases of the liver, gall-bladder, spleen and remote abscesses may also be the means of establishing peritoneal inflammation. A common source of peritoneal inflammation is the genito-urinary tract in women.

The inflammatory irritant in secondary peritonitis is infectious. The organisms which play a prominent part in peritoneal infections are the streptococcus pyogenes, the staphylococcus pyogenes aureus or albus, the bacillus coli communis and the tubercle bacillus. The ameba coli may be found in cases of amebic dysentery. Pyemic peritonitis is the result of more distant foci of suppuration. Peritonitis may also exist as the result of pleurisy through the communication of the lymph vessels.

Pathology.—In acute generalized peritonitis the peritoneal surface is hyperemic and covered with flakes of lymph. The exudate may be plastic or fibrinous, sero-fibrinous or more frequently purulent. Putrefactive decomposition may create a thin, foul smelling and sanious fluid. Offensive gases often exist as the result of intestinal perforation or the extreme decomposition of the purulent exudate. Blood may be in evidence as the result of traumatism, cancerous or tuberculous complications. The inflammatory exudate is often excessive in quantity. The intestinal changes are characterized by an inflammatory thickening of the coats, functional paralysis and an associate intestinal catarrh.

In *acute localized peritonitis* the purulent formation is limited by the rapid formation of adhesions, and thus the pus is walled off from the rest of the peritoneal cavity. The milder forms of plastic and sero-fibrinous peritonitis in their slower development may also form protective adhesions. Large abscesses may break through the circumscribed wall and escape into the general peritoneal cavity producing sudden collapse and death from generalized peritonitis.

Symptoms.—*Acute general peritonitis* is recognized by the sudden and excruciating abdominal pain which, though diffuse usually, has its maximum intensity at the point of origin. *Tenderness* is the next symptom and this is generally limited at first but soon becomes diffuse. The patient assumes the dorsal decubitus with the thighs flexed upon the abdomen and is decidedly sensitive to the slightest disturbance from motion or respiration. *Tympanites* follows as the result of paralysis of the muscular coat of the intestine. *Vomiting,* eructations or hiccough may appear. *Constipation* is a usual condition, though, in exceptional cases, diarrhea may be bothersome. The *temperature* shows a moderate elevation (102°-104° F.). In case of perforation, which is liable to occur in typhoid or appendicitis, the symptoms of collapse suddenly appear. This will be recognized by the extreme exhaustion, the cold and clammy surfaces, the frequent, small and feeble pulse, the rapid respiration, the subnormal temperature, the hippocratic expression and suppression of urine.

Should the patient survive the early symptoms, the physical signs of effusion will be presented. *Inspection* recognizes the increasing abdominal distention. *Palpation* detects the tenderness, the abdominal distention and, in some cases of perforation, the rigidity of the abdominal wall. *Percussion* gives an exaggerated tympanitic note in the beginning and dulness, which is due to effusion, over the most dependent parts later; this, however, may be obscured by the tympanitic distention; the dulness will also vary with the change of decubitus. In pronounced effusions the upward pressure may modify the normal liver, cardiac and splenic dulness. This, however, may be restored by turning the patient on the side. This obliteration will be even more marked in case of pneumo-peritonitis.

Acute Circumscribed Peritonitis.—The symptoms of this form of peritonitis are similar to those of the general form, though not so extreme in character. Pain is *circumscribed,* but not quite so severe. *Vomiting* and other gastro-intestinal symptoms are not so marked. The *fever,* however, is inclined to go higher, and partakes of the septic character. *Rigors* and other pyemic signs are usually present, though not always pronounced. The danger of general peritoneal inflammation from rupture is always present. The *localized involvement* and fluctuation are generally recognized in the physical examination. The varieties of these abscesses are termed perinephric, pelvic, subdiaphragmatic and periappendicular. Death may take place from sudden rupture or the case may pursue a subacute or chronic course with the constant danger of severe constitutional symptoms.

Diagnosis.—In making a diagnosis the previous history is of inestimable value. In the sthenic form the sudden abdominal pain, the tympany, tenderness and vomiting, will make the diagnosis comparatively clear. In the asthenic type, however, the diagnosis is not so easily made. The early symptoms are not so indicative, but the increased abdominal tension and the more profound collapse in case of a ruptured abscess will be valuable guiding signs. *Localized fluctuation,* and evidence gained by the exploring needle, establish the presence of abscess. In differentiation we must exclude several simulating diseases. *Acute intestinal obstruction* bears a close resemblance to peritonitis, particularly as the symptoms of the latter are liable to appear in its later stages. The pains, however, are more of the colicky kind and are not accompanied by so much tenderness; vomiting is often stercoraceous and projectile in character, while the obstruction is a distinct feature. In *acute enteritis* the pain is colicky and marked by exacerbations, diarrhea generally prevails, and the constitutional symptoms of peritonitis are not present. In *enteralgia* the pain is relieved by pressure, fever is absent and none of the causes which induce peritonitis is present. In *renal colic* the paroxysmal pain starts in the lumbar region, radiates down the ureters and is associated with signs of calculus, hematuria and other urinary findings. *Biliary colic* has a different location, the pain is paroxysmal and signs of jaundice appear early. *Uterine colic* is related to the menstrual period, but tubal pregnancy, with rupture, requires most painstaking study of the differentiating symptoms. *Hysteria* may, for a time, cause some confusion, but the peculiar features should settle the uncertainty after a little study of the case. *Typhoid fever* should cause no difficulty in differential diagnosis. The diagnosis of peritonitis in children presents many difficulties, though it may be commonly associated with traumatism or appendicitis.

Prognosis.—The prognosis in general peritonitis is always unfavorable. Surgery may save a number of otherwise fatal cases when it results from perforation in typhoid fever, gastric ulcer, appendicitis, cholelithiasis. Otherwise death usually occurs in a few days. In localized peritonitis there is a much better prospect. Here we may save life by prompt surgical measures directed to the focus of infection.

Treatment.—In general peritonitis we are expected to do all within our power toward the relief of symptoms. In the majority of cases, particularly in the localized form, we may obtain best results from surgical aid and this help should be sought early. *Rest in bed* is imperative. *Liquid nourishment* is all that will be tolerated and pancreatized milk in small quantities at frequent intervals is probably the best diet. If this should disturb, nutrient enemas may be resorted to. Egg albumen, meat juices and prepared foods are usually well borne. The starvation method as suggested for appendicitis may be more advisable. Saline purgatives have been recommended on the theory that they encourage the exosmosis of serum from the blood vessels of the intestine, thus relieving the peritoneal congestion and at the same time promoting rapid absorption. In this way it is possible also to limit the danger of adhesions by increasing the peristalsis. This, however, would be dangerous in case of intestinal perforation and, therefore, it is a questionable procedure inasmuch as we are not always sure of the exact condition. It is more practical to use the salines after laparotomy rather than before.

The employment of *morphin* and *opium* are sometimes necessary for relief of extreme pain, but their indiscriminate use is liable to obscure the diagnostic symptoms and at the same time interfere with nature's effort toward recovery. In hopeless cases and particularly when surgical measures are impossible, the physician may regard this as justifiable. High normal salt enemas often give good results by increasing elimination. In collapse the normal salt solution should be used subcutaneously or intravenously at a temperature of 105°-115° F.

Local treatment has been resorted to in the majority of cases, though there is a wide difference of opinion as to its efficacy. The icebag is certainly of value in the early stages for the relief of pain, though in some cases hot applications act better. These may be used in the form of hot poultices or by the application of flannels wrung out in hot water. A new cellulose-glycerin plastic dressing gives promise of good results. Leeches applied to the abdomen are advised by some authorities. Tympany is often relieved, as in typhoid fever, by the application of turpentine stupes and the high saline enema containing ten to fifteen drops of turpentine in emulsion.

When vomiting is persistent it may be alleviated by the swallowing of bits of ice, carbonated water or, better than all, by the free drinking of hot water. Remedies like *ipecac, arsenicum* and *nux vomica* may help in this respect. Should the vomiting persist after all of these expedients have been employed, the hypodermatic use of *morphin* should be tried. *Chloretone* (gr. 5-15), because of its anesthetic effect upon the gastric mucous membrane, is worthy of trial. *Cerium oxalate, cocain, menthol* and *dilute carbolic acid* may also be considered.

Internal remedies are certainly of service in this disease. *Aconite* conforms to the peculiar features of anxiety, excitement, restlessness and the temperature of the early stage. *Belladonna* is indicated here as elsewhere when the inflammation is accompanied by a rapid, full pulse with the characteristic flushing of the face and extreme local tenderness. It also meets the delirium, should that be present, and many of the subjective symptoms which attend an acute invasion.

Bryonia naturally follows these two remedies and it is most useful as the effusion begins. It is claimed that it promotes absorption and, therefore, lessens the tendency to excessive effusion. It may to a great extent relieve the sharp and lancinating pains which are always exaggerated by the slightest motion. Thirst is another symptom which calls for this remedy; the patient calls for large quantities of water as the lips, mouth and tongue are very dry. *Cantharis* is such a useful remedy in my practice when there is acute inflammation of a serous membrane that I always compare it with bryonia. Its action is expressive of intense inflammation and its symptoms are characterized by severe cutting and burning pain. *Apis* may be compared. *Mercurius corrosivus* has been lauded in a similar respect as a remedy which relieves the intensely acute inflammation of the peritoneum. It refers at the same time to many of the associate symptoms of gastric and intestinal disturbance. *Veratrum viride* probably covers more of the extreme febrile symptoms than any other remedy; it is especially indicated in infective inflammations. We may often find use for *arsenic, china, hepar sulphur, sulphur, silicea* and possibly *potassium iodid.* Typhoid symptoms will call for such remedies as *baptisia, rhus tox, echinacea* and *muriatic acid.*

CHRONIC PERITONITIS.

Chronic inflammation of the peritoneum may exist in several forms and it will be recognized as a local or diffuse process.

Chronic Localized Peritonitis.—This is a very common condition and involves not seldom the spleen, kidneys and liver, resulting in adhesions. The region of the appendix is particularly affected. As a result of the inflammation adhesions unite contiguous viscera and are frequently the cause of intestinal obstruction. Localized acute peritonitis, associated with an inflammatory disease of the organ implicated, is generally the primary cause.

General Adhesive Peritonitis.—Chronic peritonitis may exist in the diffuse form when the peritoneal surfaces are thickened and adherent; the intestinal coils also are adherent one to another. It follows acute attacks which assume a chronic form. Rheumatism is considered as a factor and traumatism may be an inciting influence.

Proliferative Peritonitis.—This is described by Osler as an excessive thickening of the peritoneal layers without much adhesion. Cirrhosis of the stomach, cecum and ascending colon are associate conditions. The liver and spleen may be adherent to the thickened peritoneum or a chronic perihepatitis or perisplenitis may develop a thick band of connective tissue about these organs and create a subsequent sclerosis. The omentum is rolled and forms a thick mass between the stomach and colon. The peritoneum is universally thickened, though it may show excessive infiltration in patches. The effusion is slight though in exceptional instances there may be some ascites. In alcoholic patients with long continued ascites, the serous surfaces are thickened and present an opaque and dead-white appearance. In case of associate cancerous growths there may be a bloody and chylous liquid exudate.

Chronic Tuberculous Peritonitis.—This is a common form of chronic peritonitis which is due to tuberculous infection. The inflammatory thickening of the peritoneum is further complicated by the deposition of tubercles and by subsequent caseous degeneration. The liquid effusion varies in accordance with the extent of the inflammation and degeneration. Hepatic cirrhosis is often an associate complication. Blood-stained effusions are characteristic of this form of chronic peritonitis—though we must differentiate the hemorrhagic peritonitis described by Virchow in which there are layers of new connective tissue, between which are areas of extravasated blood. A similar condition may exist from the use of the trochar.

Symptoms.—*Local peritonitis* may not present any marked symptoms unless there is an excessive thickening and a tumor-like swelling. This may be detected by palpation and may be attended by pain which is very intense and colicky in character. Should intestinal strangulation be caused by the fibrous bands, signs of obstruction will appear.

The *symptoms* of the *diffuse form* are also insidious and the diagnosis is frequently in doubt prior to autopsy. Alimentary disorders may be present and constipation is a common symptom, except in the tuberculous form, when diarrhea may be present. Obstructive jaundice and ascites develop whenever the adhesions cause sufficient pressure upon the common duct or portal vein. A sense of *abdominal restriction* and pain is often present. *Hectic fever* is not uncommon; this is pronounced when there is tuberculous involvement and we shall invariably find other signs of this infection. *General emaciation* and increasing exhaustion, together with many nervous phenomena, are usually observed.

The *physical signs* are not always reliable. *Inspection* reveals an irregular abdominal enlargement, unless there is great tension of the walls. Fluctuation may be detected over limited areas by *palpation.* The rolled up and shrunken omentum and the thick bands of adhesion may often be determined by palpation. *Percussion dulness* often alternates with a tympanitic sound and varies with the amount of the effusion, the peritoneal thickening and the position of the patient. Friction fremitus is sometimes detected and a resistance to percussion is invariably discovered over the area of dulness.

Prognosis.—The disease is always grave and the prognosis should be guarded. The milder cases may pursue a chronic course which terminates in a condition of extreme asthenia. While tuberculous peritonitis has heretofore been considered almost uniformly fatal, there is recent evidence that under modern treatment fully 50 per cent recover (Yeo).

Treatment.—The medical treatment should embrace many of the measures outlined under the acute form. A highly nutritious and easily digested diet, with the use of tonics and stimulants for the increasing debility, must be prescribed. Individual symptoms may call for special remedies such as the *iodids, calcareas, argentum nitricum, chlorid of gold, mercurius* and *sulphur.* The pain is sometimes relieved by local applications of heat or more particularly from the use of *belladonna* or *ichthyol cerates.* When the effusion is excessive paracentesis may be resorted to. Yeo, of London, has used with great success *iodin* or *iodoform* applied freely and persistently to the abdomen. These were also administered internally. A mixture of equal parts of iodoform ointment and cod-liver oil is rubbed thoroughly over the abdominal surface twice daily. Internally, he gave, three times daily, iodoform (gr. 1/4) and creosote (m. 1/2) combined in a pill. Inunctions of unguentum Credé have been recommended. Surgeons have performed successful laparotomies in this disease and it is hoped that a further advance in this science may give even better results.

NEW GROWTHS IN THE PERITONEUM.

Tuberculous deposits and tuberculous peritonitis may be included under the head of new growths, but have already been discussed. Cancer of the peritoneum is, therefore, the typical form which demands consideration. Primary cancer probably exists as an endothelioma. The majority of growths are secondary to a similar disease of the stomach, liver or pelvic organs. All forms of cancer are represented in a malignant peritonitis. They are usually characterized by numerous round tumors, the most of which are small, a few only being large, and are distributed throughout the peritoneum. Colloid formations often attain a very large size and are generally found in the omentum which is indurated and forms a transverse fold which may be palpated. The disease is transmitted to other parts by way of the lymph current or by continuity. It appears in later life and women seem to be affected more than men.

Symptoms.—The history of a localized cancerous involvement will be a confirming symptom, though the early signs of peritoneal invasion may not be clearly distinguished. The early evidence of *cancerous cachexia* may be depended upon, while localized *pain* and discomfort will also be suggestive. If the liquid effusion is not excessive, *nodules* may be detected by palpation. *Emaciation,* exhaustion, anemia and ascites are frequently accompanying symptoms. In the *colloid variety* a semi-solid and non-fluctuating mass may take the place of the liquid effusion. *Blood* may be found both in the cancerous and the tuberculous effusion, which necessitates microscopic confirmation and a careful study of the differentiating symptoms. *Fever* is frequently present. The abdomen always shows enlargement, but it is not always easy to tell whether it is a colloid or ascitic distention. Fibromata and lipomata may also invade the peritoneum.

Prognosis.—The prognosis is always unfavorable and treatment is only palliative.

ASCITES.

Ascites is an accumulation of serous fluid in the peritoneal cavity as the result of some obstruction to the portal circulation.

Etiology.—The *local conditions* which produce pressure upon the portal branches are quite numerous. It may arise from a compression within the liver in case of cirrhosis, abscess, chronic passive hepatitis, or it may be the result of similar pressure outside of that organ. This may be produced by enlargement of glands in the fissure, hydatids, carcinoma or organic growths in adjacent organs. Pylethrombosis, aneurysm or pressure upon the inferior vena cava are occasional causes. Chronic inflammation of the peritoneum, either of the simple, cancerous or tuberculous form, may also cause ascitic accumulation.

The *general causes* include heart disease, pulmonary cirrhosis and emphysema, Bright's disease and, in exceptional instances, leukemia, malaria, syphilis and cancer. In cases due to these causes extensive impairment of nutrition and general circulatory disturbance while serous effusion will also be in evidence in other parts of the body. *Chylous ascites* is a rare occurrence from leakage of the lacteals or from obstruction of the thoracic duct.

Pathology.—The peritoneum does not show much pathology in the average case. It may be slightly thickened and edematous and, in cases of long standing, the thickening and opacity are pronounced. In cases of peritoneal adhesions the effusion may be encapsulated. The amount of ascitic fluid is variable, often exceeding several gallons. It is clear and transparent, having a specific gravity of 1.010-1.015 and alkaline in reaction. The color will be modified by the presence of blood, bile, chyle or fat. When allowed to stand for a while the lowest layer contains leukocytes, red blood corpuscles, fat cells, endothelium and cholesterin crystals. Fibrin may lead to the formation of a clot. Albumin is commonly detected in the fluid and is evidenced by the specific gravity, the latter being higher when the amount of albumin is increased (1.015-1.024). When the effusion is due to peritonitis the percentage of albumin is usually high.

Symptoms.—The symptoms are both subjective and objective, though they may be unnoticed when the effusion is slight. The former refer to the general signs which are adduced by the primary pathologic defect and the disturbances from ascitic accumulation. The patient may complain of a sense of *weight,* fulness and distress. There may be a dragging pain in the loins, gastro-intestinal disturbances and dyspnea. Exertion and the recumbent position will aggravate all of these symptoms. When the accumulation is large, the liver and abdominal viscera are crowded upward, while the heart and lungs may be similarly displaced. As a result of this, atelectasis may be established, the apex beat displaced upward and to the left, while cardiac and respiratory embarrassment is marked. Similar pressure upon the bladder and kidneys is liable to exist when the erect position is assumed for any length of time. Syncope may take place at any stage of the ascites.

The *physical signs* are quite distinctive when the effusion is excessive. *Inspection* reveals a uniform abdominal prominence which varies, however, in accordance with the position assumed. In the dorsal decubitus there will be bulging in the flanks, while on standing or sitting the fluid seeks the lower part of the abdomen. When the patient is lying down the thoracic organs may be displaced upward, the respiration will be labored and purely thoracic in character. The distention stretches the skin, which becomes thin and shining; the cutis may rupture and give rise to reddish-blue or rosy-red stripes, which later become white and simulate the lineae albicantes of pregnancy. The umbilical depression is eliminated or everted and abdominal hernia is not uncommon. The superficial veins of the abdomen are frequently enlarged.

Palpation detects fluctuation when there is a large effusion; when the fluid is small in quantity the patient must stand in order to discover this. Palpating the adjacent organs will be a difficult task. *Percussion* gives a dull or flat sound over the fluid, though this may be, at times, modified by intestinal resonance. The dulness always varies in accordance with the patient's position. When lying down it is most evident upon the sides, while in the standing position it gravitates to the bottom of the sac. When the patient is placed upon the hands and knees a zone of dulness may be detected around the umbilicus. Intestinal adhesions and other complications may greatly modify the percussion sounds. Resonance may be detected as high as the fourth rib in the cardiac region and auscultation may reveal a basic systolic murmur.

Diagnosis.—The diagnosis rests on the detection of abdominal fluctuation and movable dulness, together with the associate evidence of the primary cause. When ascites is well established, the physical signs are convincing. Differentiations must be made as follows: In *unilocular ovarian cyst* the general health does not show an early impairment, uterine disturbances usually precede and the swelling is unilateral in the primary stage. As a cyst develops, the enlargement is irregular and the area of dulness shows a convexity toward the head of the patient. When in the recumbent position the abdomen protrudes and fluctuation will be confined to the limits of the tumor. A vaginal examination may confirm the fact that the ovary is involved. *Chronic peritonitis* furnishes a previous history of acute peritonitis. Pain and temperature are typical symptoms, the abdomen is irregularly prominent in case of effusion, fluctuation is limited and dulness is not changeable. A *distended bladder* will not be suspected after a catheter has been used, while a *dilated stomach* presents downward displacement and gastric symptoms which should be distinctive.

Prognosis.—As ascites is a symptom of some other pathological disturbance, the prognosis depends upon the primary cause. Usually it is an unfavorable sign and, though relief may be obtained by tapping, the fluid generally returns. In exceptional instances the accumulation has not recurred after the use of the trochar. When the fluid is not removed, death may occur from syncope with asphyxia and atelectasis as associate causes.

Treatment.—The first aim in treatment should be to remove the primary cause, which, however, is usually an impossibility. In cardiac and in some renal diseases we may accomplish this quite effectually, though the ascites usually recurs. In such cases diuretics are useful and these have been already mentioned in the treatment of cardiac diseases. *Diuretin* (grs. 10), *digitalis tincture* (m. 10-15) and *hydragogin,* in similar dosage, given three or four times daily, are our most useful agents in this respect. *Strychnia* is often a valuable adjuvant in connection with these remedies. Diuretic waters, a light nitrogenous diet and a complete abstinence from stimulants are absolute essentials. Purging and hot-air treatments for procuring active skin elimination are recommended by some authors, but these measures, together with extreme diuresis, are often dangerous, on account of the reactions which may follow. In my experience, with the exception of primary heart disease and its secondary nephritis, I have yet to record a pronounced success in the treatment of ascites.

Paracentesis is our most reliable, though only a palliative, measure. In cirrhosis of the liver this constitutes the only source of relief, and at best it will be only temporary. Care should be exercised not to withdraw the fluid fast enough to cause syncope, and strict asepsis should always be observed. The *internal medication* will look only to the relief of symptoms, and the possible correction of the primary cause. Remedies which apply in ascitic and edematous conditions are always to be considered. *Arsenicum* is a remedy which applies when there is a general anemia with gastro-intestinal disturbances; it is also useful when edema follows cardiac disturbances, but for the direct effusions of the peritoneum it is not applicable. In renal diseases it has a tendency to correct the scanty urine and the elimination of albumin. *Apis mellifica* refers to the edematous condition of the tissues. It is also efficient in dropsical effusion and anasarca dependent upon a systemic infection or an inflammation of the kidneys. *Apocynum* has for a long time been regarded as a valuable agent in dropsy. It is given in the tincture, decoction, or the fluid extract. Though this may increase the amount of urine excreted, I have never as yet found an appreciable reduction of an ascitic fluid by its use. My experience has been similar with *stigmata maydis* and remedies of this character.

Hellebore, cantharis, cannabis sativa and *pulsatilla* have their sphere of usefulness, particularly when there is a suppression of urine. *Cinchona, cedron* and *eupatorium* may be considered when malarial and other infections give rise to primary splenic enlargement. Remedies which have heretofore been mentioned in connection with hepatic congestions and the treatment for cirrhosis of the liver may also be applied.

DISEASES OF THE URINARY SYSTEM.

DISEASES OF THE KIDNEY.

ABNORMAL MOBILITY OF THE KIDNEY.

Normally the lower pole of the kidney, especially the right, can be palpated by the conjoined method. It is called a *movable kidney* when the upper end of the organ may be felt during deep inspiration and when it can be crowded down no lower than the umbilicus. A *floating kidney* is one which is movable to a greater degree than the movable kidney and may be palpated below the umbilicus.

Etiology.—The movable kidney is rarely congenital, being more frequently acquired when the supporting tissues are relaxed sufficiently to allow sudden physical effort or traumatism to force it out of its normal position. Women are afflicted oftener than men, as frequent pregnancies favor the abdominal relaxation, and the strain of labor may easily force the organ out of place. Tight lacing, violent straining at stool, heavy lifting, and occupations which predispose to sudden and violent exertion are alike conducive to the renal mobility. It is claimed that an abnormally long renal artery favors displacement, while emaciation, with consequent wasting of the fatty capsule, is also an occasional cause. Organic increase in the size of the organ predisposes to its displacement by the gravity of its own weight and adjacent tumors may accomplish the same by pressure. The right kidney, on account of its relation to the liver and its anatomic position, is more liable to dislocation than the left. Enteroptosis and visceroptosis are also common factors in producing these conditions. The influence of traumatism should never be forgotten in this condition.

Symptoms.—In the majority of instances no marked distress is experienced as the result of renal displacement and frequently it exists without manifest symptoms. Many cases are revealed by accidental physical findings and often the condition is never discovered until autopsy. The *direct* or *local symptoms* are often defined as a sense of heaviness or pressure in the loins or abdomen after violent exercise or standing on the feet for a long time. This dragging sensation is often reflected into the sacral region and it may assume the character of *violent* or *colicky pain*. There is rarely any complaint of pain directly over the seat of the kidney unless there is some obstruction of the ureter or congestion arises from pressure.

The *reflex symptoms* are often the most prominent signs of the displacement, especially in moderate mobility. Indigestion, vomiting, nausea, cardiac palpitation, constipation, flatulence, dysmenorrhea, irritable bladder and similar disturbances are traceable to this cause. The symptoms of appendicitis are often simulated. Pressure jaundice, gastric dilatation from pressure of the kidney upon the duodenum, and edema of the lower extremities from pressure upon the inferior vena cava are rare signs of this condition. *Dietl's crises* or severe attacks of abdominal pain with chills, nausea, vomiting, fever and collapse are not uncommon features of the symptomatology. It was Dietl's theory that these crises were due to torsion of the ureter or to twists or traction in the renal vessels and nerves on account of the extreme mobility. An acute and transitional hydronephrosis may thus develop. Pyonephrosis is a rare sequence. The urine is usually concentrated and may contain uric acid and calcium oxalate in excess. In case of enteroptosis (Glenard's disease) the gastro-intestinal and nervous symptoms may be aggravated. In all cases the nervous symptoms are often pronounced, a marked neurasthenia, hysteria or melancholia characterizing the mental features, while backache, headache, paresthesia, neuralgia and nervous dyspepsia may also be pronounced.

The *physical signs* are uncertain except those obtained by *palpation*. By the bimanual method, with the patient in a semi-recumbent position, it is possible to detect a movable kidney in the flank just below the ribs or a floating kidney in the inguinal or umbilical region. Pulsation of the renal artery is a rare physical finding. A knee-elbow position may allow more satisfactory palpation than the semi-recumbent posture. In some cases the upright position favors detection of a movable kidney.

Diagnosis.—A careful consideration of the symptoms already outlined, together with the physical examination, usually makes the diagnosis secure. A floating kidney is easily determined, but the slighter displacements are often overlooked. A *wandering spleen* is usually differentiated by the presence of the splenic notch, the absence of renal artery pulsation and the absence of a tympanitic note over the intervening colon; moreover, it is a rare affection. *Tumor of the gall-bladder* is most difficult of differentiation, as it may coexist with renal displacement, and there are so many signs common to both. When the gall-bladder is involved, jaundice is more liable to be a leading symptom, emaciation and nervous disorders are less common, the tumor is not so easily palpated and is more uniform in size. When the gall-bladder contains calculi it has a firmer feel and in these patients crepitus may be obtained. The displacement of the gall-bladder is lateral and not so marked as in case of floating kidney, and the tumor always descends with inspiration.

Prognosis and Treatment.—There is rarely any danger to life and a cure is possible by surgical procedure. Any local treatment aside from nephrorrhaphy is usually only palliative. There are, however, instances in which an operation can not be performed, and thus we are obliged to palliate to the best of our ability. Vigorous exercise and work which excites abdominal strain must be interdicted. The "rest cure" is often essential after the kidney has been restored to its normal position. Pads, binders and corsets are often serviceable and may be employed when it is not advisable to operate or the patient is opposed to it. Postural treatment and the increase of nutrition to encourage the deposit of perirenal fat are worthy of trial. In the crises of Dietl relief is obtained by the hypodermatic use of morphin and atropin and external heat.

CIRCULATORY DISORDERS OF THE KIDNEY.

Active Hyperemia.—This is a condition of acute or active congestion of the kidneys following an abnormal influx of *arterial* blood. The *etiological causes* are many. It may be a temporary state following exposure to cold, or it may attend such natural functions as menstruation. It may be the result of the ingestion of a large amount of fluids, it often follows the extirpation of one kidney or may exist in consequence of an operation on other parts of the urinary tract. It is always a condition which precedes acute inflammatory invasion of the kidneys. Cardiac hypertrophy, excessive exertion such as football demands or any condition which raises the arterial tension favors its development. The most frequent non-surgical cause is traced to infections like the eruptive fevers and poisoning by such drugs as copaiba, cantharides, turpentine, chloroform, etc. Prolonged renal congestion is liable to terminate in acute nephritis.

The *pathologic condition* is characterized by a normal or slightly enlarged kidney which has a deep-red color and is engorged with blood which flows freely upon section. The Malpighian tufts appear as dark-red points in the cortex. The characteristic feature of the morbid process is located in the renal arteries and the arteries of the Malpighian tufts.

Symptoms.—When the infections are causative factors, fever may be present. Headache, nausea and vomiting are occasional symptoms, while prostration may be pronounced. Pain in the loins, or along the line of the ureters and tenderness upon pressure are usual complaints. If the hyperemia is due to poisons like cantharides, turpentine, etc. there may be frequent micturition with urging and vesical tenesmus, or there may be complete suppression. The *urine* is dark-colored, its specific gravity is increased, blood is present sparingly in mild cases and abundantly in severe cases, with corresponding albuminuria; there may be a few small hyaline tube casts and the solids are first decreased then, later, increased. The amount of urine is at first increased and later it is deficient.

Diagnosis.—We may differentiate from *acute nephritis* by the absence of tube casts, dropsy and uremic symptoms. It may be distinguished from *passive hyperemia* of the kidney by the absence of cardiac lesions and absence of passive congestion of the liver.

Prognosis.—The prognosis is usually favorable, but, of course, much depends upon the cause. As a functional disturbance it should be a transitory disorder. Any infection which is prolonged or any irritating poison which is liable to cause suppression or induce uremia is naturally an unfavorable cause. A previous nephritic disease and a surgical operation under chloroform unfavorably predispose. Repeated attacks are liable to induce acute nephritis. Suppression of urine, convulsions and coma are always danger signs.

Treatment.—Rest in bed, warmth, a liquid diet and the drinking of a free amount of water are essentials. Hot foot baths, hot applications to the region of the kidneys, high normal salt enemas, saline laxatives and in some cases mucilaginous drinks are generally very serviceable. In the early stages copious sweating should be induced by hot-drinks, the hot corn pack and other methods of inducing a sweat. Venesection is also recommended. Otherwise the patient should be cared for as if experiencing an acute febrile disease.

The *remedies* which are of early advantage are principally *aconite, belladonna, berberis vulgaris, pulsatilla, cantharides* and *cannabis sativa.* Clifford Mitchell speaks highly of *terebinth* when there is a scanty and dark-colored urine containing blood and albumin. *Helonias* may be similarly considered. These remedies should be used in small material doses. *Lithia* is serviceable in the milder cases when the the "urine is not suppressed and contains no albumin." Five grain doses of lithium citrate, four times daily, is the best form for administration. The ethereal extract of *eucalyptus* is also recommended in drop doses every two or three hours. *Glonoin* is of service for prompt relief in cases of acute hyperemia when there is an underlying condition of chronic nephritis. This may be used (gr. 1/250-1/100) either by mouth or hypodermatically three or four times daily until there is a free passage of urine. *Arnica, hamamelis, baptisia, veratrum viride* and many other indicated remedies may be useful.

PASSIVE HYPEREMIA.

This is a chronic congestion or venous engorgement of the kidney, which is secondary to some other visceral diseases. It is also termed cyanotic induration. The *etiologic causes* are found in a general venous engorgement due particularly to chronic cardiac disease and less frequently to pulmonary or hepatic disease. A lesion of the mitral valve accompanied by failing compensation is the most frequent direct cause.

Aortic aneurysm, emphysema, fibroid phthisis, chronic adhesive pleuritis and cirrhosis of the liver are also causes. The disease may be occasionally traced to tumors, pregnancy, ascites and, more rarely, to thrombosis of the inferior vena cava or of the renal veins.

The *pathology* varies according to the duration of the congestion. In the early stage the kidneys "are enlarged, firm and of a dark bluish-red color." The capsule is generally non-adherent, the renal surface is smooth and its substance is hard. The marked changes are found in the medullary structure, the bases of the pyramids having a dark-red appearance, and the congested vessels resembling striae. In case of thrombosis, edema as well as hyperemia, may exist. Microscopic examination reveals the congested veins, the prominent Malpighian bodies, with an occasional ruptured tuft capillary and more or less distention of Bowman's capsule. The epithelium is either unchanged or only slightly cloudy and swollen or later fatty. In the older cases the kidneys become smaller, harder and darker as the result of granular atrophy. Hence, the term "cyanotic induration" has been applied. The capsule is adherent in spots, the renal surface presents wedge-shaped patches of hard tissue, the substance is fibroid, and the Malpighian bodies are atrophied within these patches. There is microscopic evidence of a thickening of the capillaries, veins and rarely the arteries, the glomeruli being occasionally atrophied. The tubular epithelium is slightly degenerated and in part detached from the basement membrane, while there is some evidence of round cell infiltration in spots. In extreme states a condition known as "hypostatic contraction" may exist.

Symptoms.—The early and prominent symptoms pertain to the primary disease, which is usually a cardiac disorder. With this there is generally some evidence of hepatic engorgement. Thus *edema* of the lower extremities is a characteristic symptom, while *dyspnea,* cardiac palpitation, a rapid and arrhythmic pulse are also attending symptoms. The cough which indicates pulmonic congestion is always present, the patient is exhausted and cyanosis is sometimes present. The *urine* is diminished, high in specific gravity and of a dark-red color. It contains a small amount of albumin, some blood corpuscles, a few hyaline casts, a few yellow or pale granular casts, seldom dark granular casts unless nephritis complicates, according to the chronicity and degree of the congestion. The uric acid is relatively increased and there is a marked sediment of urates.

Diagnosis.—As in all diseases of the kidney, a knowledge of the primary disease is necessary for the sake of diagnosis as well as treatment. In this disease the real trouble is likely to be in the heart. *Differential diagnosis* will consider *anemia of the kidneys, active hyperemia* and *primary chronic interstitial nephritis.* Clifford Mitchell has enumerated the following differential points in case of the latter: Compared with this, passive hyperemia is similar so far as the albumin and casts are concerned, but the volume of the urine is decreased, the specific gravity is high, the sediment is abundant, dropsy is an early sign, there is no high tension, there are no visual disorders or chronic uremia, cyanosis may be present and nocturnal urination is uncommon.

Prognosis.—Everything depends upon the primary cause, cardiac compensation being essential to recovery. There is a constant danger of acute intercurrent nephritic attacks, and exacerbations are always liable to occur should the patient indulge in any excess or suffer sudden exposure to cold. In such instances the albumin and blood may be increased. Suppression of the urine may take place if the heart is not properly stimulated. Chronic nephritis is the greatest complicating danger. Death may occur from cardiac paralysis.

Treatment.—The early and principal treatment should apply to the *cardiac disorder.* Without extensive repetition of the remedies which are mentioned under the subject of cardiac diseases, it is sufficient to say that cardiac compensation and renal elimination must be induced by the prompt, thorough and persistent use of such remedies as *hydragogin, digitalis, strophanthus, crategus, convallaria, apocynum, guarana* and *strychnia.* Strychnia is generally given as often as every three or four hours (gr. 1/60) and the other remedies should be given in the tincture (m. 5-10) three or four times daily, as the case may demand. When the heart needs stimulation we must not hesitate to stimulate it sufficiently or we accomplish no result.

Rest, moderate exercise and general improvement of the health are also mandatory requirements. The *diet* should include readily digestible foods which are well cooked. Foods that have bulk or excite flatulency should be omitted. The meals should come at regular hours and the patient should eat with due care for the digestive apparatus. *In case of dropsy* an exclusive milk diet is required for several weeks. Two to six ounces may be taken at each feeding, every four hours the first week, and ten to sixteen ounces per feeding during the second week. The quantity may be increased to twenty ounces four times daily during the third week. *Salines* and possibly stronger cathartics may be used regularly for a time. The morning sponge is of great importance, the patient should drink plenty of pure water and if suppression of urine takes place, a corn or alcohol sweat is indicated. If the heart is the only cause of the dropsy, the latter may be entirely overcome by the persistent use of the remedies just mentioned in full doses. *Guarana* in the fluid extract (drams 2) is often given every two to four hours, while *caffein* (grs. 2-5) may be given four times daily. *Strychnia* (1/60-1/30) hypodermatically is our most reliable remedy for quick results. *Camphor* (grs. 5) in sterile almond or olive oil may be required. The *suprarenal extract* (grs. 3-5) morning and night may be demanded when there is a relaxation of the peripheral vessels. *Ergot* (m. 5-20) should not be overlooked. *Basham's mixture* is a popular remedy with many practicians.

The indicated homeopathic remedies are *aconite, phosphorus, arnica, crategus, apocynum, digitalis, convallaria, adonis vernalis, belladonna* and *veratrum viride.*

PATHOLOGIC STATES OF THE URINE.

Hematuria.—This is characterized by the presence of blood in the urine. The renal causes include "congestions, acute inflammations, acute exacerbations of chronic nephritis, embolic hemorrhagic infarction, renal calculi and pyelitis, tuberculosis, malignant renal disease, diffuse myxangiomatous condition of the pelvic submucous tissue (Myles), actinomycosis (Israel), hydatids, traumatism and parasites (the filaria sanguinis hominis and distoma hematobium.)"

The *urinary tract* may be the origin, as the ureter may be lacerated in operation or the bladder may contain calculi or be affected by malignant tumors, cystitis or ulceration and rupture of varicose veins at the neck. The *urethra* also may be the seat of hemorrhage as a sequence of gonorrhea, calculi, parasites and traumatism. *General diseases* such as specific fevers, purpura, gout, scurvy, hemophilia, malaria and leukemia may produce hematuria. Another cause may be found in the renal congestion which follows chronic cardiac, pulmonary or hepatic disease, and Senator claims that there is such a condition as "renal hemophilia," while it is also possible that the condition is sometimes congenital and again vicarious. Drug effects from turpentine, copaiba and cantharides should not be overlooked.

Diagnosis.—This concerns the discovery of the blood and the source of the hemorrhage. Blood in the urine will vary in quantity, coagulability and disposition. A slight discoloration will indicate only a small amount in the urine, while a considerable amount may be detected in its passage or it may exist as a dark and coagulated sediment. The microscope will disclose the presence of blood corpuscles in the urine. Should the red corpuscles be associated with tube casts, we may justly suspect renal hemorrhage. In ammoniacal urine or urine with low specific gravity the corpuscles are pale and after remaining in acid urine they lose their disc-like shape. Blood always gives the chemical test for albumin.

The *source of the hemorrhage* is an important diagnostic point. *Hemorrhage from the ureter* may appear in long clots and *from the bladder* in "large, irregular bright-red blood clots." When the bladder is irrigated until the fluid returns perfectly clear and a repeated irrigation returns very red we know that the blood comes from that viscus. Prostatic hemorrhage is common after straining at stool. Urethral blood is discharged before the urine or it may be milked out. In renal hemorrhage the blood is mixed with the urine, giving a uniform "smoky" or brown color with blood casts and leukocytes. A few red blood corpuscles in concentrated urine may signify only renal congestion. In profuse renal hemorrhage clots, moulded in the renal pelvis and ureters, may appear. The hemorrhage from calculi is usually slight and appears at intervals.

A knowledge relative to the existence of chronic alcoholism, or venereal disease is always essential. The immediate and family history often point to the probable cause and the evidence of tuberculosis must always be taken into consideration.

Prognosis and Treatment.—The *prognosis* depends upon the history of the case, the extent and duration of the hemorrhage and the response to treatment. The *treatment* will include local measures so far as the seat of the trouble may be located. Internal hemostatics such as the extract of hamamelis, extract of hydrastis, fluid extract of red gum, adrenalin chlorid, gallic acid, lead acetate, calcium chlorid, ergot and opium have been used with varying success. Internal remedies may be employed from a symptomatic standpoint. In the latter sphere we shall have recourse to such remedies as *ipecac, hamamelis, cantharis, hydrastis, terebinth, geranium, millefolium, phosphorus, crotalus* and *thlaspi bursa pastoris.*

Hemoglobinuria.—This is characterized by the presence of blood pigments, especially methemoglobin, in the urine, the result of red corpuscle dissolution and the excretion of hemoglobin. The *cause of the hemolysis* is principally toxic. Irritant poisons or those of infectious diseases, particularly malaria, are the most common factors. It may be secondary to degenerations from tuberculosis and syphilis, while diseases of the blood like purpura and scurvy are occasional causes. It is rarely due to exposure to cold and violent physical exertion. *Epidemic hemoglobinuria* (Winckel's disease) has been observed in the new-born. *Paroxysmal hemoglobinuria* is a rare form without any known cause. It has been attributed to susceptibility to cold, violent physical exertion, syphilis, uricacidemia and Raynaud's disease.

Symptoms.—*Anemia* is usually a marked symptom and *jaundice* is sometimes manifest. In *paroxysmal hemoglobinuria* the attacks appear suddenly, have a short duration and tend to intermittency. Severe cases are preceded by a chill and fever. Colicky pains in the abdomen, headache, nausea and vomiting, lumbar pains, thirst, exhaustion and coma are occasionally present. Urticaria and purpura have been noticed and nephritis is claimed as an occasional result.

Diagnosis.—The urine has a reddish-brown color, a reddish-brown or a brownish-black sediment and is somewhat turbid. The specific gravity is lowered slightly and the reaction is acid. The microscope settles the difference between this and hematuria. In hemoglobinuria there are few or no red corpuscles and the few that are present are colorless and fragmentary. Brownish-black flakes of disintegrated hemoglobin are found and brown-tinged casts and epithelium are occasionally discovered. The chemical examination of the urine will reveal the presence of albumin. The blood serum has a red color on account of the dissolved hemoglobin. Hemoglobinuria is "further marked by the aplasticity of the red corpuscles, by their pallor, by poikilocytosis and by the presence of the irregular flakes of hemoglobin." The urine gives the chemical test for hemoglobin, but the microscope fails to find blood corpuscles in dense masses as in hematuria.

Prognosis and Treatment.—Much depends upon the cause. The ordinary paroxysmal form is favorable. Fatal cases have occurred as the result of malaria. The *treatment* is similar to that of acute nephritis. *Terebinth* is highly recommended and *phosphorus, crotalus* and *ferrum phos.* have also been suggested. In malarial hemoglobinuria treatment should be directed to the liver. *Quinin* apparently is not useful even when malaria is the cause and may cause death from quinin poisoning and from uremia. *Iodids* may be of service when syphilis is a factor. In the paroxysmal forms due consideration must be given to rest, quiet and external warmth.

Albuminuria.—By this term is meant the presence of albumin in the urine. The pathology and etiology relate to "the immediate escape of the normal blood elements, serum-albumin and serum-globulin from the vessels into the renal tubules." This is due to a transient or permanent disturbance of the nutrition of the epithelium of the tuft capillaries or of the cells surrounding the glomeruli. This point is, however, disputed by many, the claim being made that a physiological albuminuria may follow extreme muscular exercise, the ingestion of food rich in albumin, violent emotions, cold bathing, dyspepsia, etc. It is also held that it may be due to a nutritional disturbance of the membrana propria of the epithelium of the uriniferous tubules. The *causes* of albuminuria are many. It attends acute and chronic nephritis, renal congestions, certain toxemias, the amyloid, fatty and suppurative conditions of the kidneys and also renal tumors. It is present also in marked blood changes, it is often a feature of pregnancy and it may attend certain perversions of the nervous system. An *accidental albuminuria* attends the presence of pus or blood in the urine, it may be present in hyperpyrexia and a physiological albuminuria of transient endurance may attend the functional disturbance of the kidney. *Cyclic albuminuria* is characterized by albuminuric paroxysms which take place at times when the renal and urinary functions are quite normal. Extreme physical exercise, hearty meals and certain vocations apparently predispose. It is noticed also in old people who suffer from anemia and a dyspeptic or neurotic cause is often underlying. While these conditions are often innocent, the presence of albumin in the urine, even in minute quantity, may presage a gradual degeneration of the renal structure. In no sense is the persistent presence of albumin to be regarded as an innocent symptom.

Albumosuria.—This is a condition in which albumoses are voided in the urine. It is known to occur in septic conditions, in osteomalacia, myxedema and multiple bone tumors. Albumose is a "body more or less closely allied to peptones, globin, histon and the digestion albumoses, but it displays certain characteristics unknown to these substances." Albumosuria may occur at different periods of the day, but is usually transitory and rarely remittent. It is usually combined with serum-albuminuria, but tube casts are rarely found. Bone pain is a common symptom.

Indicanuria.—Indican may be found in normal urine in small quantities. Its excess is indicative of intestinal disorders, particularly those of the small intestine. The *pathologic* significance will be recognized when large quantities of albuminous matter are decomposed. When the caliber of the small intestine is reduced by an obstruction, there is a stagnation of its contents and decomposition takes place from bacterial action. "Thus indol, phenol and skatol are formed and the former, being absorbed and oxidized into indoxyl, finally appears in the urine in combination with potassium sulphate." It may be found also in acute peritonitis, chronic constipation, wasting diseases and cachetic conditions. Tumors which press upon the small intestines may cause an excess of indican in the urine. The *treatment* of indicanuria embraces the elimination of the products of albuminous decomposition in the intestines. For this purpose I have obtained good results with the salicylates, the sulphocarbolates and zymeoid. These should be given in doses of five to fifteen grains three or four times daily, plenty of water always being used.

Pyuria.—This term refers to the presence of pus in the urine. It is due to suppurative inflammation along the genito-urinary tract or it may appear in the urine in consequence of the rupture of an abscess into this tract. For further consideration refer to the subject of pyelitis.

Chyluria.—This refers to the presence of chyle in the urine and is either parasitic or non-parasitic in its origin. The parasitic form is observed commonly in tropical countries and is due to the obstruction of the thoracic duct or its branches by the filaria sanguinis hominis, causing thereby a rupture of the lymph vessels of the bladder or kidney. It is claimed also as a sequence of lymphatic duct injury and it is supposed to be associated with pregnancy.

The *urine* is increased in quantity and has a milky or opalescent appearance, due to the emulsified fat. A creamy film of fat may often be observed on the surface of the urine. Polyuria is a characteristic while albumin and fibrin are also present. Hematuria is usually an associate symptom when there is a parasitic cause. The findings of a microscopic examination resemble the fine granules and fat droplets of milk. Chyluria often appears intermittently and it may continue for a long time. In the non-parasitic form there is no danger as to life, but a cure is rare. The coagulation of chyle in the pelvis of the kidney may cause renal colic..

Choluria.—The presence of bile in the urine, or choluria, attends any disease in which there is an occlusion of the bile ducts. The urine varies in color from a greenish-yellow to a brown or brownish-black color, the predominating color being yellow. Albumin is often present in slight quantity and bile-stained hyaline and finely granular casts are occasionally observed. The urine is particularly foamy. Bile pigment in the urine must be differentiated from the yellow urine caused by such drugs as santonin and rhubarb. These do not respond to the tests for bile and there is usually no yellow foam.

Urobilinuria.—This is a condition manifested by the presence of urobilin in appreciable quantities in the urine. It gives the urine a reddish-brown color and is seen most frequently in fevers and hepatic diseases. It may appear following the resorption of effusions; the hemorrhagic diathesis, purpura, pernicious anemia and acute chlorosis may be causative factors. *Urobilinicterus* is a discoloration of the skin caused by the deposit of urobilin in the tissues.

Glycosuria.—By this term is meant the presence of sugar (glucose) in the urine and it is always a question whether this is a sign of disease or whether it is dependent upon the excessive use of saccharine or starchy foods. The most natural suspicion excited by the appearance of sugar in the urine is that we have a case of diabetes mellitus. This, however, is not always true, as explained under that subject. Many of the infectious diseases may give an intermittent or paroxysmal glycosuria while a disease of the nervous system which excites the floor of the fourth ventricle may likewise induce it. It is also well known that mental excitement—grief, worry, shock, etc., often develop a temporary glycosuria, while traumatism and operations upon the brain are occasional factors in its appearance. Convulsions, meningitis, hemorrhages and concussion are liable to incite a temporary elimination of sugar which has no marked significance. Pregnancy, such drugs as morphin, phloridzin and chloroform, obesity, pancreatic disease, exophthalmic goitre and several other diseases have been known temporarily to disturb the glycolytic function, and thereby induce the passage of sugar in the urine. The use of alcohol, carbohydrates or glucose in excess may often explain its presence. In simple glycosuria the urine may be only slightly changed from the normal and its presence may not be suspected without chemical examination.

Acetonuria, Diaceticaciduria and β-oxybutyricaciduria.—These three conditions are almost inseparable and are closely associated with glycosuria and diabetic coma. *Acetonuria* may be present, in slight degree, in normal conditions. It is also observed in autointoxication, diabetes, carcinoma, fevers and some nervous diseases. *Diaceticaciduria and β-oxybutyricaciduria* are always pathologic expressions, being associated with acetonuria in diabetic coma and rarely found in fever. Von Jaksch claims that they are expressive of an independent disease. It is now held that β-oxybutyric acid is the direct cause of diabetic coma.

Lithuria.—This condition refers to the presence of an excess of uric acid and urates in the urine. Many causes may be given which predispose to this condition, the "lithic acid" or gouty diathesis being most prominent. Rheumatism, fever, leukemia, pulmonary and other diseases, as well as an excessive nitrogenous diet are mentioned as predisposing factors. A temporary high acidity and a deficiency in mineral salts may diminish the solubility of uric acid and, therefore, favor the condition of lithuria. The urine has a high specific gravity, a dark-red color and an acid reaction. The uric acid will deposit, when the urine is allowed to stand, in a bright, cayenne pepper-like formation. According to the theory of various German observers, the nature of this diseased condition is an excessive nuclein-catabolism. The *clinical features* may be enumerated as follows: Mental irritability, depression and exhaustion; headaches, insomnia and restlessness at night; occasional neuralgic pains here and there, often over the symphysis pubis, backache and gouty phenomena.

In the *treatment* of this condition the patient will be required to abstain from intoxicants of all kinds and vigorous outdoor exercise must be regularly instituted. Baths, well-ventilated sleeping apartments, massage, hydrotherapy, electricity and the tonics are often essential. An abundance of alkaline water must be consumed and the patient should constantly avoid the heavier proteids and the use of any article of food which is in the least stimulating. Meat should be eaten sparingly, though, as Croftan says, the "increased nutrition and resisting powers that accrue from an animal diet are essential." A change of climate is often advantageous to the successful treatment and the alkaline waters of many of our springs are conducive to a favorable result if freely and properly used. Laxatives such as Carlsbad salt, sodium phosphate, Thialion salt, Hunyadi and Rubinat waters and many of the well known salines are also efficacious and should be used in a systematic manner.

Remedies should be prescribed according to their indications. *Bryonia, cimicifuga, cedron, pulsatilla, colchicin, agaricin* and *mercurius* are often indicated for the wandering pains. The *lithium compounds* have been extensively employed, though it is questionable how much good is obtained from these. *Lithium benzoate* (gr. 2-10) three or four times daily, is recommended by Clifford Mitchell, the object being to dissolve the sediment of urates and to increase the urea-uric acid ratio. *Nux vomica, ignatia, arsenicum, iris versicolor, chelidonium* and *cocculus* are often indicated when there are gastric and hepatic disorders. *Nitro-muriatic acid* dilute and *hydrochloric acid* dilute (m. 5-10) in one-half glass of water after meals, are also useful in chronic gastritis or tardy digestion. The *glycero-phosphates* may be prescribed when there is a low urea-uric acid ratio and a high ratio of urea to phosphoric acid. When anemia is a factor, as it often is, *arsenic, iron* and many of the standard iron preparations may be employed to advantage.

Oxaluria.—This term is employed with reference to the persistent excess of calcium oxalate in the urine. The association of nervous symptoms, indigestion, jaundice and spermatorrhea is frequently observed. Certain articles of food, such as plums, strawberries, tomatoes, apples, bananas and rhubarb favor the formation of oxalate crystals. The precipitation of these crystals is due to the absence of sodium phosphate which keeps them in solution. Their presence in the urine does not always imply an increased excretion of this salt. They appear most commonly as "minute, regular, highly-refracting octahedra or, more rarely, as hour-glass and dumb-bell shaped crystals."

The *etiology* and *pathology* of oxaluria has been considered by some as a distinct disease characterized, aside from the urinary findings, by the symptoms of neurasthenia, dyspepsia and hypochondriasis. It has also been accounted for as a disturbance of metabolism particularly of the fats and carbohydrates. It is found also in wasting diseases like diabetes mellitus, tuberculosis and cancer. It has also been noticed in paresis of the insane, catarrhal jaundice and calculi. Sediments of urates or uric acid frequently show the oxalate in large quantity. The greatest immediate danger from the presence of these crystals is the formation of a calculus, while nephritis from prolonged irritation is a remote possibility.

The *treatment* should include a change of climate for the betterment of the general health and vigorous attention to the diet and elimination. The *diet* should exclude those articles of food which are rich in oxalic acid, as previously mentioned, and sweets should also be restricted. Distilled water should be used freely. Meat should not be used in excess. *Remedies* which have been mentioned by experienced practicians are *nitro-hydrochloric acid* (m. 5-10) in water three or four times daily after meals. *Basham's mixture, lithiated hydrangea, lithiated sorghum* and *lysidin* are frequently used. *Oxalic acid, picric acid, kali sulph., kali carb., senna* and *berberis vulgaris* have been advocated as frequently indicated homeopathic remedies.

Phosphaturia.—The voiding of urine containing an excess of phosphates occurs under various conditions. Excess of earthy phosphates is not of any serious consequence as far as known. The excess of the phosphoric acid combined with the alkalies may be of some serious moment. Indigestion established by hyperchlorhydria and food or drinks which are abundant in alkaline salts are also causes. There is an increase of phosphates in wasting diseases like tuberculosis, leukemia and chronic articular rheumatism.

The *clinical features* are generally characterized by a pronounced mental and physical exhaustion, sexual weakness and ill defined pains in the back and limbs. The patient is also inclined to be neurasthenic and many times a state of hypochondriasis intervenes. In a majority of cases there are no well defined or positive symptoms which are indicative of phosphaturia. The voiding of urine containing a sediment of phosphates is a constant feature of phosphaturia.

The *treatment* of this condition should be directed, first of all, to dietetic and hygienic improvement. Hard water should be avoided and a light but nutritious diet should be adopted. Rest and a proper amount of exercise are essentials for the sake of the exhausted nervous system. Among the remedies we may mention *hypophosphites, glycero-phosphates, arsenicum, berberis aquifolium, kali mur.,* and *phosphoric acid.* Other remedies which relate to the predisposing causes are always to be used according to their indications.

Cystinuria.—This refers to the rare condition of an excess of cystin in the urine. While the causes are not well understood, it is usually noticed after infectious diseases, rheumatic fever being the most common cause. Syphilis is also regarded as a possible cause. Hereditary influences are also supposed to be important factors, while a disturbed metabolism as seen in gout, obesity and allied conditions may establish it. Brieger has referred to the relation of ptomains to cystinuria, and it is likely that these may account for it in many instances.

UREMIA.

This term is employed to define a condition manifested by stupor, coma, convulsions or paralysis together with gastro-intestinal disturbances.

Etiology and Pathology.—The true cause of uremia is not understood. It is generally held that the condition is due to diminished renal activity and the consequent retention of metabolic products. It usually arises from renal disease. The theory entertained by Traube to the effect that the symptoms of uremia are due to acute cerebral edema with anemia may account for some cases. The theory of the retention of effete material also fails fully to explain the disease, inasmuch as a complete ligation of the renal arteries and ureters in animals does not establish uremia. The injection of urea and healthy urine into the circulation also fails to cause uremia. Impaired renal activity does not sufficiently explain these symptoms. The claim that the kidney has an internal secretion which governs metabolism is refuted because ligation of the renal vessels does not result in uremia. A more recent and more plausible claim is to the effect that a poison is developed in the body as the result of nephritis. Whether this arises from the development of a micro-organism or depends upon perverted metabolism has not yet been determined. Lazarus-Barlow says "the symptoms of uremia are not immediately due to renal condition, but to the disorganization of the hepatic function of forming urea from ammonium carbamate, which the renal disease brings about owing, perhaps, to the absence of an internal secretion. Ammonium carbamate, therefore, collects in the blood and when a certain degree of fermentation has been produced it acts upon the brain with the result that convulsions, coma, etc., supervene." Those interested in the theoretical considerations of uremia are referred to the experiments of Hahn, Massen, Nencki and Pawlow.

Symptoms.—In the *common form* of uremia the patient becomes drowsy and passes into coma which may be preceded by delirium. In this condition there is some twitching of the muscles. A *convulsive type* is a more severe manifestation of acute uremia. In this form the patient is suddenly seized with an epileptoid attack, the muscular twitchings and convulsions beginning in the face or fingers and soon spreading over the entire body. One attack rapidly follows another or the patient may remain in a state which simulates the "status epilepticus." Unconsciousness is the distinctive feature and should there be intervals of paroxysmal relief mental impairment will be noticed. The temperature usually falls, the knee-jerk is greatly exaggerated and the pupils are contracted. This form of uremia is most commonly observed in the toxemic eclampsia of pregnant women.

The *respiratory form* is characterized by marked dyspnea. In this form asthma is simulated and a peculiar feature is the socalled "expiratory hiss." Cyanosis is present only to a slight degree unless there is an attending cardiac disorder. There is usually no mental perversion and the patient complains constantly of the difficult breathing. In aggravated cases the "Cheyne-Stokes" respiration is apparent, but here it is not such a serious sign as it is in other diseases. The *maniacal form* of uremia is fortunately not so common. In this the patient is extremely restless, excited and often violent. Drowsiness finally ensues and the patient passes into a terminal coma.

A *paralytic form* is of occasional occurrence. This is characterized by hemiplegia or a suddenly developed monoplegia, the paralysis taking place as the result of an intoxication of the nerve centers controlling the muscles involved. Cerebral apoplexy may occur at the same time. *Another form* of uremia characterized by *continuous insomnia* is occasionally met with. Associated with this there is usually pronounced muscular irritability or cramps and a persistent hiccough. Often the uremia is attended by violent *gastro-intestinal disturbances,* vomiting, intense nausea, and sometimes purging.

Uremic amaurosis is the feature of some cases. In this there is a sudden development of blindness which is usually bilateral. The amaurosis, in favorable cases, lasts only for a few days and in many cases only a few hours. The vision is liable to return as suddenly as it disappeared and the prognosis is invariably favorable as to the return of vision. There is no pronounced retinal change excepting an edematous condition. Acute nephritis following eruptive fevers is the most frequent precursor of this form of uremia.

The *skin* of the patient is usually pale in uremic coma and pruritus may sometimes attend. The *peculiar odor* of the uremic breath is very characteristic and may at times be ammoniacal. The *dropsy* attending an existing renal disease is usually increased, though in many instances it may show a marked reduction when the uremic symptoms appear.

Diagnosis and Prognosis.—When uremia assumes an acute form the coma and convulsions do not last long, as death usually comes within 24 to 72 hours. The chronic form may continue longer with symptoms of dyspnea, nausea, and muscular twitchings lasting intermittently for weeks. Albuminuria, with tube casts, arteriosclerosis and accentuation of the aortic second sound and the other symptoms above described are sufficient to make a diagnosis. While uremia is always a grave condition it is not necessarily fatal, as it may often be overcome by prompt and vigorous treatment. *Uremic monoplegia* and *hemiplegia* caused by the rupture of a blood vessel, or by an embolus or thrombus must always be differentiated. In *cerebral hemorrhage* the pupils are dilated or unequal, and the stertorous breathing, the hemiplegia, the sudden and more complete loss of consciousness, the persistent slow pulse, the atheromatous condition of the arteries and negative urinary findings are the chief symptoms which differentiate it from uremia. In *alcoholic narcosis* the pupils are generally contracted, there is no stertorous breathing, the odor of alcohol is detected in the breath, paralysis is not in evidence, complete unconsciousness rarely exists, the pulse is frequent and feeble and there are no convulsions. We may observe a combination of true uremia and alcoholic narcosis. In *opium poisoning* the pupils are contracted and there is often some evidence of drug taking. In *meningitis* the character of the onset, the rigidity of the neck, the photophobia, the fever, the mild delirium and Kernig's sign will guide in making the differentiation. *Diabetic coma, typhoid asthenia* and *acute miliary tuberculosis* must be determined according to the characteristic symptoms outlined under the discussion of those diseases.

Treatment.—This is taken up to a considerable extent under the treatment of diseases of the kidney. The prompt elimination of the poison which creates it is of first importance and this must be accomplished or helped by catharsis, diuresis, diaphoresis and, if necessary, by venesection. The early administration of a saline purge, like citrate of magnesia, or a more vigorous cathartic like calomel (grs. 1-3) should be early resorted to. The high normal salt enema in large quantities (quarts 1-2) and repeated if it is not retained is usually a good emergency measure. To encourage copious perspiration this should be used at as high a temperature as practicable (105°-115° F.). The intravenous and subcutaneous introduction of this is still preferred by many experienced practicians. In case of profound weakness and collapse the alcohol sweat, the corn sweat, or the establishment of prompt and copious perspiration by the application of outside heat is usually efficacious for temporary relief at least. In all of these the bed must be prepared so that the patient rests between woolen blankets and has an abundance of outer covering. The ice bag or a cool cloth should be kept constantly on the head during this procedure and the patient should, if not unconscious, be given bits of ice frequently or sips of cool water. Lumbar puncture has been recommended. *Pilocarpin* (gr. 1/12-1/8) hypodermatically will often start elimination when other measures fail. The heart must be constantly watched and in case of failure *nitroglycerin* (grs. 1/200-1/100) should be given hypodermatically every three or four hours. *Strychnia* (gr. 1/60-1/30) given in the same manner is often efficacious. Pulmonary edema is often a serious sign and should always be guarded against, *strychnia* and *atropin* (gr. 1/150-1/100) usually being the best emergency remedies in these conditions. Remedies and specific treatment are referred to under the heading of acute nephritis.

AMYLOID DISEASE OF THE KIDNEYS.

Amyloid or waxy degeneration of the kidneys is usually a feature of a similar process affecting other organs. The renal change is characterized by the "deposit of lardacein in the subintimal stratum of the blood vessels, in the glomeruli and, to a lesser degree in the connective tissue of the tubules." (Hare.)

Etiology.—A suppurative process of long standing or any prolonged toxemia is liable to produce this condition. Suppuration of bone tissue as observed in Pott's disease and hip-joint disease is a common factor. Tuberculosis of the lungs with cavity formation, or of the intestine is often the primary disease. The changes incident to a chronic diffuse parenchymatous nephritis may be considered as etiologic possibilities, while syphilis and malaria are frequently the predisposing diseases. Gout, leukemia, cancer and rarely chronic endocarditis have also been regarded as factors.

Pathology.—The kidneys are enlarged and on section present a polished appearance. If there is connective tissue increase the kidneys may be small. The cortex is pale in color while the pyramids have a deep-red color, the glomeruli being markedly visible. An aqueous solution of iodin applied to the cut surface of the areas undergoing amyloid change gives a deep mahogany-red color; if a dilute aqueous solution of sulphuric acid is applied this brown hue is changed to blue. A microscopical examination will reveal the fact that the tufts in the capsules, being mostly affected, are changed into a waxy mass. The "disease also involves the afferent and efferent blood vessels of the tuft and the straight or direct vessels of the kidney." The connective tissue of the tubules is increased, the epithelial cells are often granular or fatty and in advanced cases much of the secretory structure becomes atrophied. Other organs, particularly the liver and spleen, reveal a similar pathologic condition. The heart always suffers an hypertrophy if it does not partake of the amyloid change.

Symptoms.—The symptoms vary according to the degree of the amyloid degeneration. Often no distinctive symptoms are in evidence other than the *peculiar pallor* which affects the skin. *Dropsy,* to a slight degree, may be recognized and symptoms of *cardiac enfeeblement* prevail in the majority of advanced cases. *Diarrhea* is sometimes present as a result of amyloid infiltration of the intestines. The *urine* is voided freely, is clear and has a low specific gravity, while *albumin* is generally present in large quantities, globulin being increased out of proportion to the serum-albumin. *Waxy casts* are in evidence, though fatty and hyaline may also be present. Other symptoms of nephritis may be present.

Prognosis.—The prognosis depends upon the gravity of the primary cause and the degree of the pathologic change in the kidney. Other organs are liable to succumb to the amyloid change before the kidneys. When the casts and albumin are abundant and there is evidence of wasting and exhaustion a fatal termination may be expected.

Treatment.—Little may be expected from remedies or specific treatment in well advanced cases. The treatment should apply to the primary cause so far as possible. Hygienic measures, tonics and the iodids have been advised. *Phosphorus* is probably our best indicated homeopathic remedy. If a septic focus is the underlying factor surgical measures are indicated.

NEPHROLITHIASIS.

This term relates to the formation of calculi in the kidney substance or renal pelvis, the concretions being composed of solids derived from the urine.

Etiology and Pathology.—These concretions vary decidedly in size and character, renal sand, gravel or stone existing under different circumstances. The *larger calculi* may completely fill the renal pelvis, project into the ureter and crowd upon the calices and infundibula. The smaller calculi are often voided with the urine or become engaged in their passage through the ureter thus causing excruciating pain and obstruction to the free passage of urine. The formations may invade the renal tissues also. The tips of the pyramids may be irritated by the formation of uric acid after birth and cause much suffering in the early days of child-life. Sodium and ammonium urates are also found at the tips of the pyramids in adults when gout is a factor and lime-salts may be deposited in the same part of the kidney in elderly people.

Several varieties of these concretions are formed from the urinary solids and become foreign bodies of pathologic character when there is an abnormal tendency to crystallization and an albuminoid substance is present to hold the particles together. The *uric acid calculi* are of most common occurrence. They are found frequently in old people with scanty urine or with those whose urine is diminished on account of abnormal perspiration. "As a consequence of concentration and high activity of the urine, the uric acid and urates are readily separated in solid form and held together by the albuminous matrix." Such stones have a reddish appearance, are very hard and their surface is smooth.

Phosphatic calculi occur more frequently in the bladder than in the kidney. They are composed of calcium phosphate, ammonio-magnesium phosphate or both and are the result of bladder disease when the urine is ammoniacal.

Calcium oxalate concretions are of rare occurrence in the kidney forming the so-called mulberry calculi. They often develop about a nucleus of uric acid, are very hard, rough and are dark in color. When these exist there is usually an abundance of oxalate in the urine and digestive or metabolic disorders are at fault.

Other concretions composed of *cystin, xanthin, carbonate* of *lime, fatty or saponaceous matters, indigo* and *fibrin* are of rare occurrence.

Secondary lesions or the sequelae of nephrolithiasis are characterized usually by a pyelitis which may be either catarrhal, diphtheritic or purulent. Often the stone may exist without the knowledge of the patient unless some accident causes injury of the kidney structure and then hematuria takes place. Should the stone be dislodged renal colic ensues. Pyelonephritis or paranephric abscess may be sequelae in severe cases. Hydronephrosis may likewise exist when the ureter is closed by a calculus, pressure-necrosis and perforation occasionally following this.

The *causes* of these abnormal formations are not well understood. In advanced life the uric acid form is most frequently observed. Men are apparently predisposed more than women, gout, excessive meat eating, and the sedentary life invariably having a favorable influence. Heredity is considered a factor in some cases. In certain parts of the country the mineral ingredients of the drinking water may account for the prevalence of such cases. The precipitation of insoluble ingredients in excess, as the result of changes in the urine, is a condition favorable to the formation of renal calculi. The real factor, however, is the presence of some substance in the urinary tract which acts as a nucleus for these formations. A highly acid urine, an excess of uric acid, a deficiency of salines and of normal urinary coloring matter are also favorable factors.

Symptoms.—When a calculus causes any trouble it is manifested by *renal colic.* This occurs (1) when the dislodged calculus blocks the ureter and causes an obstruction to the urinary flow and consequent distention; or (2) when the pressure of the urine on the stone forces it down into the ureter; or (3) when the walls of the ureter spasmodically contract on account of the stone's presence. The latter is probably more frequent than the other two conditions. The *pain* is tearing in character and occurs in agonizing spasms which respond to nothing but anodynes. It starts from the lumbar region and extends down the ureter into the groin and often into the genitalia. The paroxysms may have temporary remissions but are liable to recur until the calculus has passed the point of impingement. The patient usually presents the signs of collapse, perspiration is marked, the pulse is small, rapid and feeble, nervous symptoms are pronounced and the convulsions may end in complete syncope. *Fever* is not generally present except when pyelitis develops and then chills will exist. The *urine* is scanty, suppressed or bloody and frequent attempts at micturition are made. In case of pyelitis, pus and pelvic epithelium may be found in the urine. The results of the mechanical irritation are reflected in the sequelae.

Chronic nephrolithiasis may exist for years without marked symptomatology, though in some cases the renal colic may recur at various intervals. During the interparoxysmal stage the patient may enjoy good health with the exception of frequent and irritating urination and the passage of minute uric-acid crystals. Pain in the region of the imbedded stone is liable to exist. In cases of long standing hematuria or at least smoky urine is observed. Sometimes a renal *intermittent fever* is detected. The general health of the patient is, however, usually very good. Gastric disturbances, headache and other evidences of renal trouble may be in evidence.

Diagnosis.—The renal colic with the peculiar radiation of pain into the groin, genitalia and the inside of the thigh is characteristic diagnostic feature. The systemic and renal conditions which predispose must also be taken into account and these will only be understood after careful and frequent physical and urinary examinations. When a concretion is found in the voided urine the colicky pains are explained. Differential diagnosis must be carefully made. *Biliary colic* has a pain referred to the upper abdominal zone and the radiation is toward the back. There is no urinary disturbance, hematuria is not present while jaundice, gastric disturbance, the characteristic stools with the possible presence of gall-stones and other hepatic symptoms usually settle the diagnosis. *Vesical calculi* cause some confusion in diagnosis, but these give rise to a pain located in the bladder region and not limited to one side. With vesical calculi the urine is usually alkaline, while with renal calculi the urine is acid. *Appendicular* colic may simulate renal colic on the right side but the latter is more paroxysmal, less uniform in location, rigidity is not observed and the other classical signs of appendicitis are usually absent. *Neuralgia* of the lumbo-abdominal form will often closely simulate renal colic but the hematuria and other urinary symptoms are absent. *Pleurisy* will be differentiated by the localized pain, the fixation of the diaphragm and the friction sound in many cases. *Renal tuberculosis* will require careful study to interpret the meaning of hematuria and pyuria. *Intestinal colic* is not so sharply defined as renal colic and does not present the same urinary features or the retraction of the testicle. At present the Roentgen rays are of some service in locating the tumor. The diagnosis of renal calculus by means of ureteral catheterization should be applied if possible. The scratch-marks, if obtained, will furnish positive evidence if precautions are taken in the operation.

Prognosis.—An attack of renal colic may be fatal but it is rarely so. The prognosis should, however, be guarded in every case for the disease may run a chronic course, which greatly undermines the patient's health. The complications are also liable to be serious. If a surgical operation is possible we may expect relief. A large calculus remaining in the kidney is liable to induce pyelonephritis, hydronephrosis, paranephric abscess, nephritis and uremia.

Treatment.—*Prevention* of the formation of calculi should in every way be studied. The habits of the patient should be regulated and the diet should exclude rich foods and particularly the heavy meats. Vegetables and fruits should be eaten freely. Clifford Mitchell calls attention to the error of beef eating and beer drinking, particularly after exercise and profuse perspiration. Pure, soft water in abundance should be a part of the simple dietary. Massage, consistent physical exercise, sufficient rest and recreation, the daily bath or cool shower and every measure which develops a healthy physical resistance should be a part of the general treatment. Alcohol should not be permitted, though some authors claim that whiskey may be allowed. Sweet wines and beer should be interdicted.

To diminish the *size of the stone* should be a primary object in the treatment. To do this requires, so far as possible, a knowledge of the character of the sediment. *Uric acid* is dissolved by alkalies and the object should be to alkalize the urine as much as possible. When there is a *phosphatic stone* this is contraindicated. *Calcium oxalate* is insoluble in both acids and alkalies but as it is produced under the same conditions as the uric acid sediments the treatment may be the same. The free use of *alkaline mineral waters* for the solution of uric acid is of service. These are obtained at many of the popular springs. In this country we have many waters of the kind on the market. Londonderry, Buffalo, Geneva, Waukesha and Saratoga waters are well known and there are also many other good ones. These may be made more active by the addition of alkaline salts such as the citrate of potassium and the bicarbonate of potassium and lithia, say five to ten grains to each glass of water. It is not best to allow the patient to flood the system with these waters, one quart every twenty-four hours being sufficient for the average case. *Liquor potassae* (U. S. P.) will overcome the acid tendency. It may be given (1-2 c.c.) in milk at suitable intervals. *Piperazin* (grs. 5) two or three times daily, is of evident value, though this is disputed by some. *Lycetal, dimethyltartrate,* (grs. 3-5) four times daily is also recommended. *Lysidin* (m. 10 of a 50 per cent solution) may be used, largely diluted with water, three times daily. The solution of the *oxalate calculi* calls for similar treatment. *Calcium carbonate* (grs. 10-20) three or four times daily is highly recommended by Von Noorden, Croftan and others.

The *phosphatic formations* are not so easily managed, as our means for acidifying the urine are limited. The mineral acids, though they do not always obtain the result desired, may be used to counteract the high degree of alkalinity. In these cases the object desired is attained, in a limited degree, by the use of *benzoic acid* and *boric acid.* Five to ten grains of each may be given three or four times daily if the stomach is not disturbed. Diluent drinks, particularly distilled water, may be used freely. When the character of the stone is unknown the neutral mineral waters should be used. Even in uric acid conditions the too free use of alkaline waters may encourage the deposit of phosphates and, in some instances, stones may exist with alternate layers of uric acid and phosphatic depositions. When the urine is alkaline and cloudy from phosphatic deposits *urotropin,* in five grain doses, may be used three or four times daily with benefit.

The treatment of *renal colic* will tax the physician's capabilities to afford the patient relief. Often this is not obtained until the stone is dislodged. *Morphin* hypodermatically administered (gr. 1/4-1/2) is invariably demanded though it sometimes fails to relieve. A *hot bath* may give greater relief. *Nitroglycerin* hypodermatically (gr. 1/200-1/100) may mitigate the pain somewhat, as it relaxes the spasm. *Atropin* (gr. 1/100) and *hyoscin* (1/200) together hypodermatically will often cause such relaxation as to permit the stone to pass through the ureter or more reasonably, to fall back into the pelvis of the kidney. *Chloroform,* if the heart will endure it, may give temporary suppression of pain. High enemas of hot normal salt solution are always indicated. Clifford Mitchell advises the internal use of glycerin (1-4 ounces); this he claims will shorten the attack, relieve the pain and possibly prevent its repetition. Such specific remedies as *uva ursi, vesicaria, belladonna, hyoscyamus, codein, sweet spirit of nitre, buchu, belladonna, pulsatilla* and *stigmata maydis* have been recommended. Hemorrhage may demand *ergot* (m. 5-30), *alum* (grs. 10-15), *gallic acid* (grs. 10-20), *hamamelis, geranium* or *trillium* in material doses.

The *surgical treatment* is often demanded and in serious cases when relief is possible in this way the patient should not be allowed to suffer without such interference. In repeated attacks with evidence of pyelitis, surgery should not be delayed. In simple cases a nephrotomy or nephro-lithotomy may be performed but when the kidney is badly damaged by the stone nephrectomy may be required.

The *symptomatic treatment* of nephrolithiasis may, according to Clifford Mitchell, call for the following remedies: *Berberis,* when the renal colic is attended by sharp and stitching pains in the renal region with red sediment in the urine; *argentum nitricum,* when there is a dull aching pain across the back and extending to the bladder, the urine being acid and containing uric acid; *cantharis* for the pain and congestion during the passage of calculi; *coccus cacti* for vesical tenesmus, lancinating pains and red sand in the urine; *cannabis* with urging to urinate (*camphor, apis* and *copaiba* may be compared); *dioscorea* with pains shooting into the testicle and lower extremities (*colocynth, chamomilla* and *bryonia* may be compared); *nitric acid* in oxalate calculi; *nux vomica* with painful and ineffectual desire to urinate and tearing pains at the neck of the bladder; *phosphorus* when the urine is brown and contains red sand, with smarting and burning in the urethra; *sarsaparilla* with frequent, scanty and painless discharge of urine; *sepia* with turbid urine and constant desire to urinate; *pareira* (m. 3-5) when the renal colic starts.

ACUTE NEPHRITIS.

This term is used to cover that class of cases characterized by an acute diffuse inflammation of the kidneys. It has been referred to as acute Bright's disease and may be mild or severe in form. It often borders closely upon acute hyperemia which has already been described.

Etiology.—Acute nephritis may occur at any time of life, though it is more inclined to affect those who have not reached middle life. Males are more frequently predisposed on account of exposure to cold and the more irregular and intemperate life. The free or excessive use of alcohol is also a factor.

The *exciting causes* first include those which affect the skin. This refers not only to skin diseases or affections of the skin like burns, but it may also include any condition like exposure to cold or alcoholism which affects the normal function of the skin. A preëxisting perversion of renal function may also attend this sudden disturbance of skin function. *Toxic agents* most naturally favor the disease and among these we may give precedence to the acute infections. Scarlet fever has been regarded as the most common precursor of the disease, but any infectious disease may be the inciting cause. Among the other causative infectious diseases we may mention typhoid, influenza, pneumonia, diphtheria, measles, chicken-pox, septicopyemia, meningitis, rheumatic fever and tuberculosis; syphilis and malaria apparently are not common causes. The nephritis may occur as a primary disease. *Chemical toxic agents* may induce the nephritis by a direct irritation. Common irritants of this kind are turpentine, cantharides, carbolic and salicylic acids, mineral acids, potassium chlorate, phosphorus, lead, arsenic, mercury, etc. Under this head may also be included the highly spiced and adulterated foods, alcoholic excess and the irritations which may arise from anesthetics. *Pregnancy* which produces renal engorgement by mechanical pressure is also a frequent factor in acute nephritis. The nutritional changes and the blood condition which prevails at this time are also favorable to the development of the disease. *Traumatism* has been considered as a cause and without doubt it has its influence, but how great this may be is a matter of conjecture. Some acute attacks may be dependent upon a latent form of chronic nephritis.

Pathology.—The pathologic manifestations depend, to a great extent, upon the previous condition of the kidneys, the character of the irritating cause and the severity of the inflammatory invasion. Mild cases may display only slight anatomic changes. The microscopic appearance is usually characterized by the enlarged, swollen and somewhat softened kidney. These findings depend largely upon the extent of the inflammatory edema and the interstitial exudation. In appearance the kidney is usually red and will present a bloody character upon section. In other instances it is pale and mottled. When congestion is pronounced it is possible to discover a hemorrhage beneath the capsule, or hyperemic patches on the outer and cut surfaces may alternate with the opaque and whitish portions. The cortex is hyperemic or deeply mottled in severe cases or only slightly congested in mild cases. The pyramids are generally intensely red, the surfaces are smooth and the capsule is not adherent.

The *microscopical changes* will reveal a cloudy swelling or a granular degeneration of the epithelium of the Malpighian tufts, Bowman's capsule and of the cortical uriniferous tubules, when there are exudative changes in the interstitial tissue. The acute parenchymatous changes may be limited to the glomeruli. "The cells are swollen, opaque and irregular in shape while the cell contents are granular. A further advance in the process is seen in cellular coagulation, necrosis or disintegration, desquamation of the cells and hyaline degeneration of masses of them in the tubules. Acute degenerative changes are frequently found in the acute infectious diseases or when inorganic poisons have been introduced into the body."

Fatty degeneration may take place in case of phosphorus poisoning and a rapid cell-necrosis may be incident to very severe cases.

The *characteristic changes* are not found alone in the renal parenchyma but the inflammatory exudate is also discovered between the tubules, in the tubules and the Malpighian bodies. The tubules may be dilated and "choked with degenerated cells or hyaline casts." Different pathologic stages are usually in evidence in various parts of the kidney. In some parts these may be only a slight cellular infiltration of the intertubular tissues; in other parts desquamated epithelial cells, hyaline casts in the tubules and interstitial exudate are in evidence. The epithelium of the glomeruli, particularly in the tuft capillaries, is swollen and opaque while the capillaries are not normal in shape and size. The function of the glomeruli is not generally lost, as the epithelium is usually soon restored. In the acute cases the intertubular new tissue is generally cellular while in the subacute cases it is more fibrous in character. In the extreme cases of acute nephritis anasarca, peritoneal, pericardial and pleural dropsy are probable while many complicating conditions may arise.

Symptoms.—The symptoms are usually characterized by a *sudden onset* which often appears in connection with an infectious disease or the sudden exposure to cold. A chill or chilliness may be followed by a fever which is not usually high. Nausea, pain in the back and other reflex disturbances may announce the onset. In children a convulsion of uremic character is not uncommon. The *urine* is generally scanty or suppressed and an *edematous puffiness* about the eyes and on the face is attended by a peculiar pallor of the skin due to the rapidly developing anemia. *Swelling about the ankles,* which develops into a general anasarca in extreme cases, is often noticed. The scrotum and penis are often involved. When the edema is particularly marked, a rapid effusion of fluid may take place into the peritoneal cavity or possibly into the pleural spaces, after which pulmonary edema will develop rapidly. Prostration is generally pronounced but in mild cases the disease may be overlooked, unless the urine is examined.

The *characteristics of the urine* must be taken into consideration. The quantity is greatly reduced, the specific gravity is increased in the early stage, the color is unusually dark, varying according to the amount of blood voided. The urea and gross solids are diminished, the reaction is acid and a large precipitate of albumin will be found upon boiling the urine. The microscopic examination will reveal the presence of blood, renal epithelium, hyaline, epithelial and leukocyte casts and granular debris.

The *pulse* is often hard, tense, and, in the later stages of an extreme case, very rapid. This depends upon the cardiac condition. In grave cases *acute cardiac dilatation* may take place and this, together with the pulmonary congestion, leads often to a sudden death. In the average case with any gravity the high arterial tension, the accentuated aortic second sound and a feeble first sound at the apex will be recognized. *Uremic manifestations* may occur at any time.

In the *milder cases* which are secondary to any infection except scarlet fever, albuminuria, dropsy, uremia and anemia are not so marked. A slight amount of albumin and a few hyaline casts are all that are clinically expressive of the disease except the mild subjective symptoms. A typhoid condition may follow a degenerative nephritis incident to acute toxemia. The nephritis of pregnancy, though always a constant danger, may be characterized only by its gradual onset and the presence of a slight amount of albumin and a few hyaline casts. "In that form of acute or productive nephritis in which there is a tendency to the formation of patches or wedges of fibrous tissue" high fever, cerebral and circulatory disturbances, aggravated urinary findings, extreme dropsy, dyspnea, vomiting and diarrhea may precede the convulsions and coma which end in death. One danger of an acute nephritis is its termination in the chronic form from the lack of proper care and treatment.

Diagnosis.—Every case which shows a pallor of the skin, puffy eyelids and prostration should cause us to suspect acute nephritis. The careful examination of the urine will always make the diagnosis certain. In acute diffuse nephritis, headache, restlessness, muscular twitchings, nausea and vomiting, a tense pulse, slight fever, dyspnea, dropsy and anemia will be confirmative symptoms. The constant presence of albumin, tube-casts, red and white corpuscles in the urine, together with a diminished amount of dark colored urine will usually explain the cause of illness, particularly when the history of the case is known.

Prognosis.—The ordinary case of nephritis, resulting from such slight causes as exposure to cold has a duration of a few days to three or four weeks. The albuminuria gradually diminishes, the casts disappear by degrees, the daily excretion of urea is increased, the amount of urine also increases and the acute symptoms soon subside under appropriate care and treatment. The prognosis depends largely upon the severity of the renal inflammation and the primary cause. *Scarlatinal nephritis* is usually regarded as serious and the same is true when the disease follows many of the more severe infections like yellow fever, cholera, etc. *Phosphorus, mercurial or arsenical poisoning* also may cause an incurable nephritis by the extensive necrosis of renal epithelium. Acute nephritis which attends such infectious diseases as typhoid fever, pneumonia and diphtheria frequently responds readily to treatment. In these cases when the urinary symptoms improve, the dropsy gradually subsides, the anemia fades away and the patient begins to take on a normal appearance. In some instances the dropsy may subside, while the albumin, in slight amount, persists for some time. Rarely the acute affection terminates in a chronic parenchymatous nephritis. A serious phase of the disease is apparent when uremia, with cerebral manifestations, coma and convulsions, is recognized. Severe and persistent edema and such complications as pleuritis, pneumonia, peritonitis, meningitis, hydrothorax, and cardiac disease are always to be feared. Suppression of the urine is also an unfavorable sign.

Treatment.—In the management of acute nephritis it is necessary to give much attention to the primary disease but the direct treatment must relate to the condition of the kidney alone. The *first object* of *treatment* should be to restore the normal secretory function and, therefore, we are often obliged to resort to diaphoretics and cathartics to force the skin and the bowels to perform the elimination which the kidneys temporarily fail to do. Care should be exercised not to crowd this feature of treatment beyond the proper requirement. Powerful diuretics are not demanded in the average case of acute nephritis. Simple diluent drinks and a mild cathartic in conjunction with the indicated remedy are often sufficient in this respect. Distilled water may be taken for this purpose. Many of the popular "mineral waters" which are essentially pure spring water may be freely prescribed, the object being to increase the elimination of urine to normal or a little above the normal standard. It is not wise for the patient to drink an abnormal amount of water, particularly when dropsical symptoms are present, to the extent that the kidney is overtaxed in its eliminating function. Colon flushing, followed by the rectal injection of a pint of normal salt solution, may be demanded in uremic states. Active purgation is not always required but in case of extreme constipation it is demanded. This supplements the kidney function to a certain extent. When hepatic and intestinal relief are attained in this manner there is less toxin absorption and the action of remedies will be more perfect. Should it be necessary to obtain more active catharsis I know of no better remedy for this purpose than elaterin (grs. 1/20-1/10) three or four times daily until a free watery movement is obtained. Then the remedy may be gradually dispensed with. Hot baths, or better, tepid baths, may be given daily or twice daily, providing great care is exercised lest the patient be chilled. Friction should be thoroughly applied during and after the bath and the patient should be well protected with bed clothing when placed in bed after the bath. Some patients will not endure this exposure and in such instances the daily tepid sponge may be sufficient in this respect.

The *diet* is of great importance in treatment for no food should be allowed which will irritate the kidneys or increase the toxin supply. Therefore the amount of proteids should be reduced to the minimum. Milk is allowable in most cases, but should not be used when the urine is very scanty or suppressed. Even during convalescence the proteids should be resumed with greatest care according to the exigency of the case. One to two quarts may be given at regular and frequent intervals in the average case. It is best to boil the milk and it may be diluted with Vichy water and a little lime water may be added. Clifford Mitchell advises the use of arrow-root gruel and claims that this should be the only diet when the urine is "nearly or wholly suppressed." When improvement is established he advises the use of fresh buttermilk, milk-gruel with rice, flour-gruel, malted milk, Kumyss, Kazol, barley-water and oatmeal-water. In the later stages of convalescence he recommends clear vegetable soups, light broths from pigeon, fowl or veal and also hen's eggs in moderation if they agree. No red meat extracts should be permitted. Two or three weeks later when recovery is evidently established a light diet of solid food may be given with caution. Fruit juices may be freely prescribed. If albumin has not disappeared or is not decidedly decreasing even this diet should not be allowed. It is a safe procedure to continue the milk and gruel diet until all danger signs have disappeared. If *thirst* is *extreme* and the urine is not scanty or the dropsy increasing, a large amount of water may be taken, and it is well to have this hot. Should nausea and vomiting be troublesome, the liquid food and the water may be given in smaller quantities but more frequently.

The old method of *cupping* is not a feature of modern treatment though it may be indicated in some cases. The dietetic, hygienic and saline treatment are usually sufficient as local measures. Leeching is also abandoned by present-day practicians and venesection is only required when cardiac conditions demand it. The hot wet-pack, the steam-bath and the hot-air sweat may be required in some cases. The alcohol-sweat and the corn-sweat, described elsewhere, are also indicated in some cases. Aspiration may be required in conditions of hydrothorax, hydro-pericardium or ascites, while the excessive edema may demand the relief of multiple skin incisions in the ankles.

When the nephritis has persisted for some time and the daily amount of excreted urine is not increased, it may be necessary to use, in addition to the water, some of the standard diuretics. In this line we shall have use for *potassium bitartrate* or *acetate* (grs. 10-20) three or four times daily, *sodium benzoate* (grs. 10-15) or the more prompt acting diuretics, *diuretin* (grs. 10), or *agurin* (grs. 8-15) every three hours. Should the heart, by its weakness, become a factor in the suppression of the urine it will be necessary to use some of the cardiac stimulants like *digitalis tincture* (m. 5-10) or the infusion (dram 1) every three or four hours or in severe cases such a remedy as *hydragogin* (m. 10-15) every three hours may be demanded. Other remedies which are applicable in cardiac failure at this time are *convallaria* (m. 5-10), *adonis* *vernalis* (m. 10-15), *strophanthus* (m. 5-10), *crategus* (m. 10-15), *caffein citrate* (grs. 5-10) and *Basham's mixture* (dram 1) three or four times daily. This heroic treatment is rarely required and is only mentioned for use in case of necessity.

Complete rest in bed is of greatest importance and is an early requirement. The room should be warm and the patient should be well covered to encourage a free but not depressing perspiration. Too much freedom in this respect is often permitted and serious results have followed not keeping the patient constantly in bed and under strict observation. After the bath the patient should be put back in bed, covered with plenty of blankets and with a warm water bag at the feet. Perspiration may be encouraged by the "alcohol sweat" in severe cases. For this the patient is placed between woolen blankets, hot bricks are saturated with alcohol, wrapped in woolens and placed as near the patient as possible. Hot ears of corn may be substituted for the bricks. When the patient has endured the sweat long enough to start elimination he should be rubbed dry and allowed to rest.

In case of *acute uremia* prompt and vigorous treatment is required. The wet-pack is very useful in this respect particularly in the case of children. The bed should be covered with woolen blankets, a sheet dipped in boiling water is then spread upon the bed and the child wrapped quickly in this and rolled in the blankets with the head alone protruding. An ice-bag or a cloth wet in cool water should be placed upon the head. The child should be allowed to rest quietly in this bath for some time and when it is removed a cool sponging should be given. A pint of normal salt solution may be injected into the intestine and it is well to precede this with a colon flushing if possible. The wet-pack, the introduction of normal salt solution and the colon flushing should be repeated in a few hours if the desired relief is not obtained. The colon flushing followed by the normal salt enema, should be given daily even though there are marked signs of improvement. Subcutaneous or, better, intravenous injections of a quart of normal salt solution may be given in extreme cases six to twelve hours apart. The *vapor* or *hot-air bath* are advised by many, but the latter is contraindicated when dyspnea and cardiac enfeeblement are marked symptoms. Venesection in this emergency is advised by some practicians.

Pilocarpin hydrochlorate has been employed for many years in case of this emergency. The danger incident to the use of this remedy must not be overlooked and, therefore, it is wise to use it with great caution. In fact it is far safer to rely upon other expedients. When the remedy is demanded in severe cases it may be administerd hypodermatically (grs. 1/16-1/6) and if the case is desperate it may be repeated in one to three hours. In children the remedy should be avoided if possible though rarely it may be given hypodermatically (grs. 1/20-1/16). In anticipation of uremic convulsions Clifford Mitchell advises its use as follows: "One teaspoonful of the first decimal dilution of jaborandi is put into half a glass of water and a teaspoonful dose is given every fifteen minutes until perspiration occurs; then it is to be given less frequently." The danger of cardiac depression is always to be regarded in the employment of this remedy and for that reason ***strychnia, digitalis, caffein*** and other cardiac stimulants should be promptly employed when indicated. Shattuck advises the hypodermatic use of brandy or ether in this emergency. The hot-air bath and other measures, which aid elimination, may be employed to promote the perspiration when pilocarpin is used; the heart stimulation should be vigorous when this is done. The bladder should be catheterized in every case to avoid bladder retention of urine. Catharsis, at this time, may sometimes be established by the use of croton oil "in one-half drop doses, repeated every hour" until the bowels move freely. Clifford Mitchell says "the oil may be rubbed up with a small powder of sugar of milk and placed on the patient's tongue even when he is unconscious." The saline and other diuretics should also be employed continuously at this time. Rectal injections containing acetate of potash (grs. 10-15) may be given two or three hours apart. Such cardiac stimulants as ***hydragogin, diuretin, digitalis, strophanthus, convallaria*** and *strychnia* will favor the diuresis.

The *coma* and *convulsions* will require immediate and vigorous treatment. In addition to the expedients already suggested and the remedies which are mentioned later it is usually advisable to administer *nitroglycerin* (gr. 1/50-1/100) and *strychnia* (gr. 1/60-1/30) at intervals of two or three hours as the patient may endure. Venesection is advised by many in this emergency. When the convulsive seizures are severe, chloroform inhalations may be required, though *amyl-nitrite* is often sufficient for this purpose. *Oxygen* is also serviceable in some cases. *Potassium bromid* (dram 1) and *chloral hydrate* (dram 1/2) may be given to adults, in the form of a rectal injection, to conquer the spasm. When the muscular twitchings do not respond to these measures, *hydrobromate* of *hyoscin* (gr. 1/100) or *morphin* (gr. 1/4) may be administered hypodermatically.

The *remedies* which may be administered according to the homeopathic principle are numerous. *Aconite* is usually of first service and according to our knowledge of its symptomatology is indicated in the early stages when the nervous excitement, dry surfaces, full pulse and scanty urine suggest the disease. The first decimal is usually required and some give it ten drops of the tincture in one-half glass of water, two teaspoonfuls every hour or two for a time. This heroic dosage, however, is unsafe and rarely required. *Apis mellifica* is indicated early when the facial and general edema are pronounced. Without doubt we too frequently disregard the usefulness of this remedy. It is essential always to know that we have a reliable apium virus and when this is true, the second or third decimal triturations are usually sufficient when the dose is often repeated. Aside from the rapidly developing anasarca it will be necessary to have some of the other typical symptoms of apis. The characteristic stinging sensation, the absence of thirst, the dyspnea which attends the pronounced edema, the torpid mental condition, the edematous condition in the throat together with the albuminous urine and the scanty urination are guiding symptoms. Some regard this as a better remedy for chronic nephritis than for the acute form. *Arsenicum* is closely allied to apis, though it is more useful in the subacute cases which are slow in the recovery. In this dropsy, attended by a more pronounced anemia, is a characteristic; the typical exhaustion, restlessness and thirst are apparent when this is indicated and with it there is usually some digestive disturbance. *Apocynum* may be compared with apis, though the dropsy is attended by thirst, gastric irritability and some hepatic disturbance. *Cantharis* is regarded by many as a valuable remedy in the acute stage when the burning scanty urine, tenesmus, pain in the renal region and other acute symptoms point to this remedy. It should be used not lower than the first decimal potency and generally in the third. *Belladonna* and *veratrum viride* will be required in many cases during the hyperemic stage and will be decided upon according to their suggestive symptomatology. At the subsidence of the initial stage we may find use for *rhus tox.* as typhoid symptoms may intervene. With this we shall have occasion to compare *echinacea, verbena, baptisia, bryonia* and *arnica.* When the renal condition is associated with vesical trouble *berberis vulgaris, vesicaria, benzoic acid, stigmata maydis* and *viburnum* may be indicated.

Terebinth has been highly recommended by many when the urine is scanty, bloody and symptoms of acute poisoning are present. It is, however, a remedy which should be employed with due care against its physiological action. *Copper arsenite* is recommended by Goodno in uremic conditions and *mercurius corrosivus* is advised by the same author when dropsy has subsided or has not been a prominent feature in the disease. Should there be severe headache and other signs of digestive disturbance it may be well to substitute *mercurius dulcis.* In such conditions we shall have further use for *ipecac, nux vomica, iris versicolor* or *chelidonium.* In the nervous symptoms which precede or attend the convulsion *agaricin, valerianate of ammonia, hyoscyamus, stramonium, jaborandi* and *cicuta virosa* and other indicated remedies may be employed. *Helleborus niger* is often serviceable when the dropsy is extreme, micturition is frequent and scanty and gastric disturbances are associated with pains in the head and mental torpor. Edema of the lungs will demand in addition to *nitroglycerin, atropin, digitalis, theobromin* and *diuretin* such remedies as *phosphorus, tartar emetic* and *arsenicum,* while in addition to these, gastro-intestinal edema will call for *veratrum album.* *Helonias* is a remedy which will often be indicated, especially in the acute nephritis of pregnancy, or when uterine disorders attend or complicate the disease. In this respect we shall have reason to consider *pulsatilla, aletris* and *gelsemium.*

Many other remedies which meet the special therapeutic requirements will be indicated according to the totality of their symptoms.

ACUTE INTERSTITIAL NON-SUPPURATIVE NEPHRITIS.

Councilman and others claim that this lesion exists as a special form of acute nephritis. Clifford Mitchell describes it as "an acute inflammation of the kidneys affecting principally the interstitial tissue or stroma."

Etiology.—The most of these cases are the result of acute infectious diseases in children. Scarlet fever and diphtheria are, therefore, regarded as frequent causes. Acute endocarditis, measles, pneumonia, whooping cough and cerebrospinal fever are also regarded as possible causes. The *clinical* history is not satisfactorily known. Councilman detected albuminuria, Biermier observed diminished urine, dropsy and uremia shortly before death, and Sörenson found slight edema and ascites in one case.

The *pathologic histology* is characterized by "an intense cellular infiltration in the interstitial tissue or stroma, particularly marked at the bases of the pyramids, the border zone beneath the capsule and about the glomeruli. The cells are identical with Unna's plasma cells. The tubules are widely separated by areas of interstitial tissue, and their epithelium presents various degrees of degeneration. The glomeruli show no essential changes apart from periglomerular accumulation of plasma cells." The *pathologic anatomy* shows a kidney which may be slightly enlarged but two or three times its normal weight. The capsule strips easily, the surface is pale-gray in color resembling an amyloid kidney, the veins are injected and often surrounded by small opaque nodules. Upon section there is slight contrast between the appearance of the medulla and cortex though the latter is greatly increased in width. The glomeruli are not prominent and usually are invisible. The surface of the section is gray and opaque and marked by small hyperemic and ecchymotic areas. The pyramids are darker than the cortex and these are in lines from the pyramids into the cortex. The tissue is soft, friable and moist, an opaque milky fiuid exuding upon pressure. In many of these cases there are no marked clinical features other than albuminuria.

CHRONIC NEPHRITIS.

While it is possible for classical study to divide and subdivide the forms of chronic nephritis it seems best, in a work of this kind, to concentrate them under one head which is expressive of the pathologic process and the rapidity of the inflammatory development. Several types may be included under this head, two forms, however, the subchronic or non-indurative, formerly called chronic parenchymatous nephritis, and the chronic or indurative, formerly called interstitial nephritis being prominent. These may exist simultaneously, as the inflammatory action may partake of more than one type at the same time. Hence it has been considered best by some authors to regard all forms of chronic nephritis in the sense of a diffuse nephritis the parenchyma at one time revealing most of the pathologic changes, while at other times the interstitial tissue suffers most.

Etiology.—While there are many factors which contribute to chronic nephritis we may attribute it to the *excessive use of alcohol* and the *overeating* which this induces. To this should always be added the lack of physical exercise. *Exposure* to great changes of temperature is frequently an exciting cause and it is well to add to this such factors as lead poisoning, gout, syphilis and digestive disturbances which cause autoinfections. It is possible that any of the acute infections may be the primary cause long after the infectious disease has been cured. Chronic nephritis may rarely immediately follow acute nephritis. In fact we may say that any toxic cause "acting slowly and persistently may establish an insidious nephritis."

Without doubt the disease is closely allied to *arterial changes* and in every case of arteriosclerosis we may expect to find a developing chronic nephritis. While the vascular change may, in some instances, first develop in the kidney it is safe to assume that the kidney is or will be affected when arteriosclerosis or other vascular changes exist. The relation of the heart to chronic nephritis is very intimate when the arteriosclerotic change is present. The *true etiologic cause* is many times undiscovered. Chronic *non-indurative* (parenchymatous) nephritis is generally observed in early and middle life, while the *indurative* (interstitial) is more common in advanced years, but youth is not exempt. In the latter heredity has a possible influence, while some people have a tendency toward sclerotic tissues. *Uricemia* which deranges the hepatic function may be a common contributing influence while the nervous strain which always attends "high living" is an incidental factor which must not be disregarded. Some have claimed that a cold moist climate naturally predisposes to chronic indurative nephritis (interstitial nephritis or contracted kidney). In some cases it is very difficult to draw a line of differentiation between acute and chronic nephritis. Some of the recent deductions regarding the etiology have considered the effects of traumatism which may induce a degenerative change, and malaria has been recognized as a probable factor. Dieulafoy calls attention to the close relation of toxic nephritis and appendicitis. Harrington has demonstrated satisfactorily that animals fed on borated foods revealed renal changes analogous to chronic or subacute nephritis of an indefinite type. This will of course call our attention more closely to the dangers of preserved foods which contain this drug.

The *frequency of chronic nephritis* has alarmed the profession and should attract the attention of the laity. The present tendency toward "high living" is the underlying cause, and unless people more nearly adopt simple living, particularly as regards diet, the percentage of deaths will be greatly increased. The unfortunate fact is that we rarely know of the nephritis until some infectious disease, like pneumonia, tuberculosis or typhoid afflicts the patient and the renal condition is often the cause of the death, or at least the failure to recover perfect health.

CHRONIC NON-INDURATIVE (PARENCHYMATOUS) NEPHRITIS.

This form of nephritis has been described by such terms as "chronic desquamative nephritis" on account of the desquamation of the epithelial cells which line the tubules and as "chronic tubular nephritis" inasmuch as the uriniferous tubules are involved. It has also been referred to as a "chronic glomerulo-nephritis" because the glomeruli are chiefly affected. It is better defined as chronic diffuse nephritis without induration or subchronic nephritis.

Pathology.—The *large white kidney,* one of the forms of this nephritis, has a pale or yellow color, the subcapsular veins are prominent, the surface is smooth and the capsule is easily detached. The tissue may be cut without resistance and section will reveal the fact that the enlargement is due to the broadening of the cortex. The medullary pyramids are somewhat lighter in color than in the normal kidney, but do not approach the pale yellowish hue of the cortex; in some cases the pyramids are congested. *Microscopic examination* shows "the epithelium, especially that of the convoluted tubules, granular or even fatty, desquamating and coalescing to form casts, which may be recognized in position or, having been passed, these casts leave tubules which are imperfectly lined by epithelial cells." There is usually a similar change in the glomerular epithelium and likewise in the epithelial cells of some of the pyramidal tubes, while hemorrhage may take place into the interstitial tissue.

The *large mottled kidney* represents the large white kidney plus hemorrhagic areas, the mottling being due to alternations of hemorrhagic and fatty areas. There is also a *large red kidney,* in which the mottling is not prominent.

The *secondary contracted kidney* (formerly called small white) is an advanced stage of the large non-indurative kidney, the increase of connective tissue and the resultant cicatricial contraction accounting for the change in size. Sometimes the interstitial increase does not enhance the size of the kidney. The surface is slightly granulated and the capsule is proportionately adherent. In appearance it is characterized as a pale, granular kidney, somewhat mottled by red spots. It resists incision on account of the fibrous increase. Microscopic examination reveals "extensive degeneration and disintegration of the epithelium of the glomeruli and convoluted tubules, with atrophy of the parenchyma, and a corresponding increase of the interstitial connective tissue." Foci representing fatty changes may be discovered and waxy degeneration may also attend.

The *cardiovascular changes* in non-indurative (parenchymatous) nephritis are not so marked as in indurative (interstitial) nephritis. In the secondary contracted kidney, however, arteriosclerosis and cardiac hypertrophy are more common. Retinal hemorrhages are rarely observed and the serous cavities have an excess of fluid, while edema in the subcutaneous fat is occasionally recognized.

Symptoms.—The insidious development of symptoms and their usual subacute character often obscures the diagnosis until the disease is well advanced. Close observation of the patient should discover the gradual impairment of health and the loss of flesh which is ominous. When the diffuse nephritis is well developed *edema* is in evidence. At first this may be simply a puffiness of the eyelids or a swelling of the ankles, but it soon becomes more marked in the legs and the face becomes decidedly swollen. Recumbency often changes the location of the excessive dropsy. The *greasy, pallid skin* is a characteristic expression of the anemia and toxemic condition and the "stupid facies" is equally indicative of the languor and drowsiness of which the patient complains. The *anasarca* is further characterized by pitting on pressure, and this will at first be noticed where the shoes are laced or where any pressure follows from the tightness of any garment. The edema may increase until the avoirdupois is excessive, all areas of loose subcutaneous tissue, like the scrotum, suffering from the distention. Dropsy of the serous sacs may develop and there may be a sudden edema of the larynx and lungs. *Dyspnea* now becomes a factor of the symptomatology and this is always enhanced proportionately to the toxemia and the heart's condition. The *first heart* sound loses its strength, while the *second sound* is usually accentuated. The *pulse* is rapid and the arterial tension is increased.

The *urine* in non-indurative (parenchymatous) nephritis is scanty at first, though it may become normal after a time. The specific gravity is low and albumin is present usually in large amount (0.5 per cent or more by weight). Urea is decidedly diminished, the urine is turbid in appearance and sometimes smoky-yellow, though infrequently it is macroscopically unchanged. The sediment, which is abundant, is made up of a large number of fatty, granular, hyaline and epithelial casts, leukocytes, and red blood corpuscles. In some cases the granular casts are large and black. Uric acid crystals are quite frequently found, and fat globules are often numerous. The microscopic field is remarkable for the number and variety of objects contained in it.

Uremic symptoms may appear suddenly, but these are usually preceded by nausea, vomiting and nervous excitement. Apathy and stupor are common symptoms, but coma and convulsions are the serious phases in this condition. A temporary monoplegia, aphasia or hemiplegia is not uncommon in this stage. *Albuminuric retinitis* may be discovered in some cases. The *skin* over the most edematous parts may become eczematous and trophic disturbances may be recognized. The *temperature* in chronic nephritis is usually normal unless some infectious complication exists. *Gastro-intestinal* disturbances are liable to appear independent of the uremic stage. A compensatory diarrhea is a natural result when the absorption from the edematous tissues is greater than the eliminating capacity of the kidney. The *nervous symptoms* are often of sudden importance in the symptomatology. When twitchings and fever are in evidence, it usually means that the rapid absorption of the effused fluid throws more poison into the system than the kidney can eliminate. Thus when nervous symptoms begin to develop, in conjunction with a disappearing edema or serous effusion, there is reason for alarm.

It must be remembered that the clinical symptoms of nephritis are only apparent in the well developed cases. In place of the typical signs which characterize the various forms of nephritis we may often find a combination of symptoms representing the different pathologic phases. We are misguided too frequently by the lack of early signs of nephritis; and for that reason we often prescribe for an apparent digestive, anemic, hepatic or nervous involvement when the trouble is really with the kidney. For that reason a latent chronic parenchymatous nephritis may exist undetected for a long time, until an acute congestive nephritis gives rise to sudden uremia.

In the *secondary contracted kidney*, when the interstitial fibrosis is marked, the quantity of urine increases, equaling or exceeding the normal amount. The albumin, however, decreases, the casts are not so numerous, but the cardiovascular changes will be recognized by the increased arterial tension.

Diagnosis.—The chief symptoms have already been enumerated: The puffy and pallid face, the dyspnea on slight exertion, the accentuation of the aortic second sound and the increasing anasarca during the later stages are thoroughly established. The examination of the urine yields the most positive diagnostic points. These are recognized by the decreased amount of urine, the large quantity of albumin, epithelial, granular and fatty casts, the red blood cells and the desquamated epithelium from the uriniferous tubules. The large, dark granular and fatty casts are most unfavorable signs. As the eliminated urea is usually diminished, uremic symptoms are always possible and are grave signs.

In the case of *secondary contracted* kidney the urine is increased in quantity, is lower in specific gravity, the edema is not usually so pronounced and the cardiovascular changes are more prominent.

The difference between non-indurative (parenchymatous) nephritis and indurative (interstitial) nephritis may be understood by outlining the chief symptoms of the latter. In *indurative nephritis* there is often a previous history of gout, syphilis, lead-poisoning, or nerve-strain, and the disease is more liable to occur late in life. This is not true of the non-indurative form, which is more liable to appear in early or middle life, while the cause is more liable to be acute alcoholism or an acute infection. The onset of the indurative form is more insidious, dropsy is not so prominent, arteriosclerosis is usually marked, cardiac hypertrophy and cerebral symptoms are common. The urine is of low specific gravity, excessive in quantity, the albuminuria is slight, the casts are not so numerous, anemia is not conspicuous, the uremic symptoms are generally severe. All of these symptoms contrast clearly with those of the non-indurative form. Another differentiation between the two is the fact that the indurative pursues a more chronic course, patients often living for many years under this condition.

Prognosis.—In non-indurative nephritis there is generally no hope for recovery, but the patient's life may be prolonged in comfort. The cause of death is generally uremia, dropsy and its sequences, cardiac failure or other complications. Recovery sometimes takes place in children when the disease follows an acute infection. Senator claims that the early dropsy which develops rapidly into general anasarca is fatal early, while a case suffering with a gradual and irregular development of dropsy may endure longer. The milder cases, therefore, may have an indefinite continuance, until suddenly the disease assumes a more classical form and death soon appears.

Treatment.—There is so much of importance in the treatment of chronic nephritis that it is well to speak first regarding the *care of the general health.* Too many patients have been allowed a dangerous freedom in this respect or at least the physician has not given sufficient warning to cause them to be apprehensive. *Exposure to cold* is a frequent cause of an aggravation in the inflammatory action, and to disregard the danger of a slight cold too often invites a sudden death. To keep the patient warm at all times often overtaxes the ingenuity of the physician, and to accomplish this it is a wise procedure to keep our patients in bed a good share of the time, or at least to keep them indoors when the weather is inclement. An *equable climate* should be sought if possible and a sufficient amount of clothing should always be worn to keep the body perfectly warm. *Overexertion* should always be avoided; a sufficient amount of rest should be demanded; stimulating drinks and tobacco should be given up, and every habit which lessens the physical resistence should be overcome. In other words, the patient must cultivate and develop a better condition of health, without which the efficiency of our treatment is lost. While we can hardly hope to restore the kidneys to their normal condition, we may so care for the patient that they may perform their limited function without any aggravation of the disease. With this object in view there is a broad field of opportunity for the physician, and we should by no means allow ourselves to regard the word nephritis with the fear that causes us to abandon all hope in treatment. The value of fresh air and sunshine should always be kept in mind.

Bathing is a feature of the treatment which should receive more than ordinary consideration, for the skin must help the kidneys to eliminate the normal fluids and solids from the body, to say nothing of the toxins which may, at times, poison the system. There is a great difference of opinion as to the choice of warm or cold baths. The warm bath is undoubtedly most efficient and less dangerous if it is taken at night with due precaution against taking cold. It is equally true that a cold sponge, with a proper amount of friction, will add to the healthy action of the skin and, therefore, favor a more constant though less excessive skin elimination. Those patients who are accustomed to the cold shower will endure it best and are less liable to take cold. To encourage sweating a patient should be well covered in bed after the bath and this should be followed by complete rest for some time. It is not best to allow a patient suffering from chronic nephritis to take a bath without going to bed after it. *Turkish baths* may be permitted under the most strict attention and only when the heart is sufficiently strong to endure the depression which follows. Should a patient suffer a chill following any bath the condition of nephritis will invariably be aggravated. The *hot-pack* has been advised by many and, if properly given as advised under the treatment of acute nephritis, it is sure to give much relief. When the urine is suppressed and dropsy is marked, this is a wise procedure; in many instances the alcohol sweat or the corn sweat may be substituted. There is a tendency to overdo the "sweating" process in the treatment of chronic nephritis. While this is to be borne in mind, yet in case there is suppression of urine and excessive edema the chief object is to relieve the system of the surplus water in circulation which the kidneys can not eliminate. More or less urea is also eliminated by this method.

The use of *water internally* is a factor of treatment which requires much thought. The value of this agent can not be denied, for its aid to elimination and value as an article of diet are well known. That it should be used ad libitum or forced to the extent which was once deemed advisable is not advised by modern therapeutists. Much depends upon the condition of the vascular system and it should always be our object to spare the heart and kidneys from excessive functional activity. Copious draughts of water engorge the vessels and thus increase the labor of the heart and excite the kidneys. Therefore only a sufficiency of pure water should be permitted and when active symptoms are present three pints of fluid are considered to be enough. Edsall and others claim that "excessive water drinking increases nitrogenous metabolism and, as the kidneys in nephritis are unable fully to deal with the products of normal metabolism, it would seem evident that excessive quantities of water must be harmful." It should not be understood from this statement that the use of water is not a valuable expedient in the treatment of chronic nephritis, but it should be recognized that careful use of it is best. Unless this feature is explained to the patient the tendency will be to deluge the system with water beyond the ability of the eliminating function to care for it. All liquids should be used with discrimination and, though some advise the use of alcohol, tea and coffee in moderation, I do not believe that they should be allowed. It is impossible to permit such indulgences with any hope that moderation will be properly observed by the patient.

The *diet* is a matter of great importance in the treatment of this disease but in this we are often inclined to extremes which are not always justifiable. That *nitrogenous food* must be avoided or at least limited is a general rule to be accepted. Much, however, depends upon the physical requirements and the renal condition. In the *severe cases* the exclusive milk diet is still our safest food in a majority of cases. It should not be indiscriminately prescribed and its prolonged usage should be regulated. Clifford Mitchell advises the administration of skimmed milk (ounces 2-6 at a time) three or four times daily at regular intervals. The amount is gradually increased (ounces 60-80 total) during the second to the fifth week. After this time, in the majority of cases, the milk should be reduced to three feedings daily when vegetables and farinaceous foods such as oatmeal, cracked wheat, granola, etc., may be gradually and carefully substituted. It is possible also to permit the use of a small amount of broiled or roasted meat daily at the noon meal. It will often be necessary to peptonize the milk, and to add ice to avoid gastric disturbance. Kumyss, buttermilk or Matzoon may take the place of milk and it is always more palatable when diluted with French Vichy and many other waters. An exclusive milk diet *should be abandoned* when there is excessive or persistent polyuria, when anemia and exhaustion are evident and when the albumin is small in amount. Should dropsy be extreme it may be advisable to hold as closely as possible to a diet of crackers, zwieback with a small amount of skimmed milk, buttermilk or Matzoon.

Many experienced practicians object decidedly to the strict employment of skimmed milk because of the enormous quantity which is essential for the necessary nourishment. Another objection is urged on account of the lack of iron in milk. Others oppose this diet alone inasmuch as the quantity of proteid in a large amount of milk is more than would be found in a limited mixed diet. Aside from the overwork of the kidneys, which is instituted by a large quantity of skimmed milk, large quantities of urea and phosphates must be eliminated by those organs, which are already impaired in their function. It is argued, therefore, that cream may be added, when the quantity of milk is reduced, the fat thus increasing the number of calories. For the same reason starchy foods may be employed, thus obtaining a necessary nourishment without exciting the renal function with an excess of fluid. It is also probable that a little meat, eggs, sugar, butter, toast, zwieback, rice and fresh vegetables may be safely added. *Condiments* and every article of food which has a tendency to irritate the kidneys must be avoided. It should not be inferred from this statement that proteids are to be used with great liberty, for it is always best to confine the diet to farinaceous foods, fish and possibly fruits, while beef tea and the strong soups are to be always carefully avoided in severe cases.

Mineral waters are advertised with unnecessary prominence, though many of them are serviceable when taken with proper limitation. When diuresis is desired many of these may be prescribed and often may be taken hot with more favorable results. The pure spring water or, better, distilled water, are usually sufficient.

Remedies in the form of drugs are not to be employed unless special conditions demand their use. When the dropsy, dyspnea, and circulatory enfeeblement suggest the necessity of cardiac stimulants we may be justified in using *hydragogin, digitalis, convallaria, adonis vernalis, strophanthus, spartein, caffein, cactus and glonoin* as advised under the treatment of cardiac diseases. Uremia, hematuria, persistent albuminuria and other associate conditions have been referred to under their proper headings. Gastro-intestinal and hepatic disorders will always require attention according to the symptom-complex. *Diaphoresis* is generally sufficiently established by the hot baths, vapor baths, hot packs or in extreme cases by the use of pilocarpin, as suggested in the treatment of acute nephritis. *Catharsis* is always an essential of treatment and this is usually obtained by the use ot standard salines, or in severe cases by means of cascara, calomel or elaterin (gr. 1/20-1/10), as previously described. On general principles it is not advisable to use powerful hydragogues unless necessity demands them. In many instances the use of ox-gall and remedies like *chionanthus, carduus, chelidonium, iris versicolor* and *chinoidin* are sufficient for the average case. The normal salt enema is often an adjuvant to catharsis or, in some instances, it may supplant this principle of treatment.

Under the *special therapeutics* we must give more than ordinary attention to the use of *hydragogin* when dropsy and cardiac weakness are factors. This remedy contains digitalis and strophanthus tincture together with scillipicrin, scillitoxin and oxysaponin, the latter greatly aiding the diuretic action of the other remedies. It may be given in ten to fifteen drop doses three or four times daily. In my experience it is not only the best diuretic which I have ever used, but in a great many cases it has surely reduced in number or removed the casts from the urine. When there is an organic cardiac lesion or when the cardiac action is decidedly weakened I have often found it necessary to support this remedy by the alternate use of *strychnia* (gr. 1/60-1/40) three times daily, or by the adjuvant use of *adonis vernalis* (m. 10-15) with equal frequency. In extreme cases it may be well to seek the conjoint aid of *diuretin* (grs. 10) or *potassium acetate* (grs. 10-60) two to four times daily. *Apocynum* is also a valuable remedy in relieving the edema and dropsy, but its tendency to disturb the stomach often forbids its employment. Usually it may be used in the tincture (m. 15-30) or the distilled extract may be prescribed. Fluid extract of *stigmata maydis* or corn silk (m. 20-30) is a favorite remedy with some practicians. Macy's diuretic mixture, which contains equal parts of the tincture of apis, apocynum and hellebore, has been highly recommended. Basham's mixture may also be mentioned. Cream of tartar (grs. 30-60) may be prescribed in lemonade two or three times daily when prompt diuresis is desired. When the dyspnea is an extreme feature in cardiac weakness *strychnia nit.* (1/100-1/40) is often a useful remedy and it should be given hypodermatically every three or four hours until there is positive evidence of relief in this respect. *Aspidospermin* (gr. 1/100) is favorably mentioned for this purpose by many experienced practicians. Tincture of *passiflora* (m. 10-60) is also recommended in these emergencies. The cardiac stimulants, previously mentioned, generally suffice for this symptom of cardiac weakness. It is also observed that, in crises of this kind, the dyspnea is greatly relieved by any measure which encourages free elimination.

Multiple puncture of the skin in the region of the ankles is often a last resort to relieve the extreme dropsy. I do not, however, employ this nearly so much as in the past, for I find that this is not generally necessary when such cardiac stimulants as digitalis, strophanthus or hydragogin are employed in full doses. *Paracentesis abdominis* is required when all diuretic remedies fail to relieve the dropsy. I have found, however, as in the dropsy attending cirrhosis of the liver, that when this method is once begun it must be repeated at more frequent intervals.

Surgical procedure in the treatment of chronic nephritis has received much attention of late. I have not as yet been convinced by any reliable statistics that this gives any permanent relief. It is possible that the serious signs of nephritis, persisting under careful medical treatment, may demand this interference. In such operations the capsule is generally split and stripped from the kidney for some distance or the exploratory excision may be extended into the renal substance as far as the pelvis. Numerous punctures, followed by careful drainage, have been advised by prominent surgeons. It is claimed that these operations have been followed by a marked decrease in the amount of albumin and other signs of relief. It is possible that this procedure may, in the future, be of more service than it apparently is now.

The *homeopathic remedies* may be used, not so much for the cure of the nephritis, but to meet the clinical picture of the case. In the treatment of this or any other disease we are able to correct the pathologic condition by remedies only as we use them according to the suggestion of symptoms. Remedies alone will not cure this disease any more than diet and other measures alone will do it, but a conjoint use of all these therapeutic aids are essential to the treatment. In the earlier stages of this form of chronic nephritis many of the remedies used in the acute form may be continued; *cantharis, apis, mercurius corrosivus* and *arsenicum* still being useful.

As the chronic phase of the disease becomes well established other remedies may be indicated. Thus we may refer, first of all, to *ferrum phos.*, which is essentially indicated for the anemia which is now thoroughly developed. Iodin may be used in the form of *ferrum iodid,* while in many respects *calcarea iodid* is more beneficial. Iron is the one remedy which all practicians consider of value in this stage of the disease, and it may be used in many forms. Basham's mixture (acetate of ammonium and iron), Boudreaux's syrup, Blaud's mass, Hemaboloids, Hensel's iron, iron peptonate and many other preparations have been popular prescriptions. It is highly probable that *ferrum phos.* in the third decimal potency, if continued for some time, will assure more satisfactory results. *Arsenicum alb.* or *arsenicum iodid* will be indicated in many cases when the exhaustion, anemia, restlessness, gastric disturbances and burning surfaces are present. The arsenic may also be combined with bromid (arsenicum bromatum) or with hemoglobin, while many favor drop doses of Fowler's solution gradually increased to five or ten drop doses three or four times daily. *Phosphorus, phosphoric acid, nitric acid, aurum muriaticum, mezereum* and *argentum nitricum* are valuable remedies when properly indicated. *Potassium iodid, strontium iodid* and the iodids of *mercury* are very useful remedies when there is evidence of a specific history or a general tissue disintegration. In the use of *potassium iodid,* in such instances, it is absolutely necessary to gradually reach the maximum dose (grs. 20-60) and to give the remedy only after meals with an abundance of water. *Helonias,* on account of its direct action upon the kidneys and the uterus, is a very useful remedy when women are afflicted. While it is more frequently demanded in the acute stage of nephritis, it is nevertheless of use, when anemia or chlorosis are present, in the chronic stage. With this it is necessary to compare such remedies as *aletris, pulsatilla, viburnum, caulophyllum* and *cimicifuga.* When the urine is suppressed or scanty, dark colored and heavy with sediment we should naturally think of *hellebore,* which may be used in many chronic cases. With this we may compare *benzoic acid, berberis, terebinth, vesicaria, pichi, cannabis sativa* and *chimaphila.* When hematuria is a factor there are many remedies, in addition to the ones already mentioned, which may be demanded on account of their peculiar and prominent symptoms. Among these we may mention *millefolium, erigeron, acalypha indica, erecthites, ipecac, hamamelis, geranium* and *uva ursi.*

CHRONIC INDURATIVE (INTERSTITIAL) NEPHRITIS.

This form of kidney disease is also known as chronic interstitial nephritis, cirrhotic kidney, granular kidney, contracted kidney, and it is also defined by other terms which characterize the pathologic change. The disease is a chronic, diffuse nephritis which results in a contracted kidney with extensive destruction of the tubular structure and an overgrowth of interstitial connective tissue. Cardiovascular changes are invariably present.

Etiology.—While many cases of indurative nephritis reveal no cause, it is possible to discover the primary affection or the predisposing factor. *Gout* is so frequently associated with its development that it has been customary for some to define it as a gouty kidney. Lithemia and nervous dyspepsia are no doubt allied conditions which favor it and some have regarded articular rheumatism as a predisposing factor. Intestinal disturbances are found in nearly every case. The *uric acid diathesis*, or the lithemic state which generally ensues in that class of patients who overeat and overindulge in wines are without doubt commonly allied conditions. *Lead-poisoning* has also been ascribed as a cause, as many cases occur among those who work in lead. *Syphilis* and probably other acute infections favor the pathologic changes which create the contracted kidney, and it is also incident to arteriosclerosis. *Age* is a natural factor, as it is one of the natural tissue changes of old age, and hence the "senile kidney" is not an incorrect term for the disease. Allbutt attributed many cases to long-continued anxiety or great grief, and it is highly probable that the excessive strain which is put upon the nervous system by the high pressure of modern business is often of great influence in the development of the disease. *Heredity* has been given as a frequent cause, and this probably has more weight in the etiological history than we are accustomed to give it. *Prolonged passive renal congestion,* stone in the kidney, pregnancy, heart disease and many other causes have been enumerated, all of which no doubt have a direct or indirect influence in its development.

Pathology.—Some authors have defined three forms of indurative nephritis, the primary, secondary and the arteriosclerotic. The pathologic phase is, however, essentially a sclerosis, though there may be a combination of fibroid and the non-indurative (parenchymatous) form, in which the overgrowth of connective tissue is the characteristic feature. In the typical interstitial kidney the increase of fibrous tissue, which extends throughout the organ, causes a contraction and shrinkage in size and a puckering of the surface. The capsule is thick, opaque and firmly adherent, so that decapsulation tears the renal tissue. The cortex surface is roughened by round granules and occasionally small cysts may be present. The structure is firm, dense and resistant to the knife. The cut surface reveals an atrophied cortex with alternating reddish and pale parts. The pyramids also show some signs of atrophy and the color is darker than that of the cortex. *Microscopic examination* reveals the increase and irregular distribution of connective tissue, particularly in the cortex and interlobular vessels. In advanced cases the glomeruli are quite small and may be converted into fibrous bulbs. The walls of the tuft vessels may be thickened and the larger blood vessels are similarly affected. The tubules also show changes, the connective tissue often obliterating the lumen. The fibrous increase may also cause localized constriction of the tubules with resultant dilatation in other parts. The epithelium lining these tubules undergoes granular, fatty or waxy degeneration, while the tubes contain granular or fatty debris and casts. The latter are not so abundant as in the non-indurative form. Arteriosclerosis and cardiac hypertrophy are marked pathologic conditions incident to indurative nephritis. Whether these are the result of the nephritis or whether nephritis follows from these complications is not as yet settled satisfactorily. That arteriosclerosis is liable to cause cardiac hypertrophy by circulatory resistance can not be denied, but that arteriosclerosis is a pathologic factor in indurative nephritis is also well established. It is evident, therefore, that renal sclerosis, arteriosclerosis and cardiac hypertrophy without valvular disease are related conditions, all of which must receive attention in treatment.

Symptoms.—The *early symptoms* of this disease are not usually detected, and it is only in the advanced stages that our attention is called to it. It occurs in adult or later life, though it has been found in children with a history of congenital syphilis. Complicating or intercurrent diseases may aggravate the pathologic condition so that the symptoms appear earlier. The disease is common among that class of persons who live a life of high nervous tension and are inclined to whip up their lagging energies by the use of stimulants. The lack of sufficient physical exercise, together with the over-abundance of food, stimulants and nerve-strain, are, therefore, the prominent predisposing causes which should suggest the disease. *Exhaustion* without apparent cause, *vertigo* or *dizziness* are early signs which should teach the physician to be apprehensive regarding this disease. *Uremia* is often the first alarming symptom, and when this appears the disease is usually well advanced. Headache, stupor, nausea and vomiting may precede or attend this. After the uremia is relieved the headache continues, the vision is imperfect, drowsiness, lassitude, digestive disturbance and frequent micturition are complaints, and the patient is subject to a return of the uremia. *Cardiac failure* is often the first symptom which causes the patient to consult his physician, who may possibly overlook the meaning of the heart weakness and the arteriosclerosis, inasmuch as he may find no albumin in the urine. *Dyspnea* which persists independent of uremia, a continuance of the drowsiness and exhaustion without any apparent cause, the progressive loss of flesh, the dry skin and the loss of color are clinical features which should not be ignored.

The *urinary symptoms* are the most diagnostic features. The daily quantity of voided urine is increased, the twenty-four hours' amount often measuring several quarts, thus leading one to suspect diabetes. Nocturnal polyuria is a significant feature, more especially in men. The urine has a light color and is clear, the specific gravity averages below 1010, though it is rarely as low as 1005. *Albumin* is present, but not in large quantity; in some instances it may be entirely absent. The *urea* is relatively and actually reduced. *Casts* are present, but not abundant; they are usually of the hyaline character, a few granular casts and leukocytes being occasionally found. In case of uremic exacerbation or during the later stages of the disease the urine may be decreased in quantity and then albumin and the number of casts may both increase.

The *circulatory disturbances* will be evinced by the physical signs of cardiac hypertrophy. A well defined *accentuation of the aortic second sound* will be present on account of the high arterial tension. The *apex beat* is displaced downward and outward. A *systolic murmur* may be heard at the apex, due to the tension at the mitral orifice caused by the high pressure in the ventricle. In case of later compensatory failure these symptoms may be replaced by the signs of cardiac enfeeblement. The *pulse* is hard and tense, the radial artery often feeling like a hard whip-cord. This will become feeble in case of failure of cardiac compensation.

The *respiratory symptoms* are not always recognized unless a pulmonary complication like pneumonia intervenes. The *difficult breathing* is often the first symptom which calls attention to the cardiac condition and this may suggest the nephritis. *Epistaxis* is a rare symptom. *Effusions* into the lung or pleural sac may cause sudden death. In case of uremia the dyspnea may develop into orthopnea, or Cheyne-Stokes breathing may attend the uremic coma.

The *skin* is dry and a slight pallor is usually detected. *Edema* is not generally present unless cardiac failure ensues. Pruritus and eczema sometimes are present and the skin usually reflects the signs of general nutritional failure.

The *nervous symptoms* are often prominent features of this form of nephritis. These may be characterized by insomnia, muscular cramps and neuralgic pains, or they may partake of the convulsions which attend the uremic condition which is always liable to appear suddenly. *Cerebral apoplexy* may first suggest the existence of contracted kidney and the hardened arteries.

Digestive disturbances are sometimes distressing symptoms in this disease. Anorexia, nausea, vomiting and other signs of gastric perversion are frequently in evidence. Uremic diarrhea is a rare symptom and it is supposed to be due to the bacillus dysenteriae.

Nephritic retinitis is often one of the earliest signs of interstitial nephritis. The patient often complains of dimness of vision previous to this discovery. Both eyes are usually affected, a partial amblyopia being detected upon ophthalmoscopic examination. In case of neuroretinitis sudden and complete blindness, uremic amaurosis, may take place in grave cases. The optic papilla "is swollen and surrounded by retinal hemorrhages or by white dots and streaks—feather splashes." Vertigo may be a complaint while tinnitus aurium and deafness are not uncommon.

Cerebral uremia is one of the dangerous contingencies and *complications* like pneumonia, bronchitis, pleuritis, pericarditis, endocarditis, laryngitis, gastritis and hepatic cirrhosis may be looked for.

Diagnosis.—Examination of the urine reveals the positive signs of the disease. We should, however, suspect it from the appearance of the prominent symptoms just enumerated. The advanced age of the patient should intimate the possibility of interstitial nephritis when there is a persistent failure of health. Headache, dyspnea, gastro-intestinal disturbances, dimness of vision, noises in the ears, dryness and itching of the skin should cause a suspicion of this disease. Mental dulness, high pulse tension, hardened arteries, cardiac hypertrophy, an accentuated aortic second sound and general circulatory failure should excite a more marked suspicion in this respect. The sudden coma, convulsions, vomiting, dyspnea, amaurosis and apoplexy will be confirming symptoms. The gouty, alcoholic or rheumatic patient beyond middle life who presents any of these symptoms should be regarded as subject to this disease even before it appears.

Prognosis.—The contracted kidney (chronic indurative nephritis) can not be cured but the existence of this disease does not necessarily mean immediate death. The condition of the heart and the blood vessels is of greater consequence. If the patient is of good habit and the disease is not well advanced when the physician is consulted, life may be prolonged for several years providing the heart and the arterial system are not greatly impaired. High arterial tension, with cardiovascular changes, favor the chances of nutritional disorder of the kidney which is not easily restored by treatment. More than this the state of the general health is a matter of importance, for constitutional disorder, from any cause, can not fail to encourage the development of the disease. *Associate infectious or specific* disease as well as advanced age naturally predispose to an early death. The nephritis is only one of the manifestations of what we term Bright's disease. The latter disease is usually referred to by the term interstitial nephritis and, therefore, we have more than the kidney to consider in prognosis and treatment. We have to consider a toxemia of intestinal and hepatic origin, which primarily affects the cardiovascular apparatus but secondarily affects the kidneys. Thus our prognosis has reference more particularly to the underlying cause—the cardiovascular disturbance—than to the pathologic state of the kidneys. The condition of the *retinal vessels* is a determining factor in the prognosis. Signs of improvement in this respect should be regarded as favorable evidence. The sudden appearance of uremia, edema, pulmonary congestion, pneumonia or acute infections are always unfavorable unless they respond to prompt treatment. With a marked increase of the arterial tension and the appearance of retinal changes, apoplexy and acute myocardial failure may be feared.

Treatment.—The treatment of chronic indurative nephritis (contracted kidney or chronic interstitial nephritis) comprehends much that has been advised under acute nephritis and the chronic non-indurative form depending, of course, upon the acute exacerbations and other conditions which simulate these forms. The object of importance is to care for the increased arterial tension which naturally directs attention to the heart, to maintain general nutrition by the carefully regulated diet and to avoid the perversion of function by overcoming the depression induced by nervousness, worry and overwork.

The *arterial tension* is to be corrected by remedies which are regarded as cardiac tonics and vasodilators. Among the first we shall obtain best service from the *tincture of digitalis* in medium doses when the heart begins to weaken. One to five drops of the tincture three or four times daily is generally sufficient for this purpose, the minimum dose being best, inasmuch as it does not cause too forcible cardiac contraction. This should be given for some time until there is evidence of a perfect compensating cardiac power. In case of cardiac failure a larger dose is required temporarily and it may be advisable to substitute or add some of the other well known cardiac tonics. *Nitroglycerin* (grs. 1/200-1/100) three or four times daily is a vasodilator which may be cautiously used with good results for some time. Many experienced practicians advise a gradual increase in the dose (grs. 1/100-1/50) until the desired effect is obtained, the patient usually tolerating this increase for some time. *Hydragogin* is one of the most useful remedies in this disease as it seems to accomplish even more than the digitalis. Its prolonged use will show its effects not only in the cardiac and vascular improvement but when casts are present it will often cause them to disappear. *Fluid extract of broom* (m. 5) four times daily is a very useful remedy, and is highly recommended by E. M.

Bruce. *Plumbum* (6x-30x) four times daily may also be beneficial. *Chlorid of gold* or *chlorid* of *gold* and *sodium* should be considered. *Hot baths* and *rest in bed* also tend to reduce the arterial tension as they favor the dilatation of the superficial blood vessels.

When *myocardial changes* are in evidence *potassium iodid* is a remedy which should not be neglected. In severe cases it may be given in heroic doses with marked results (grs. 10-40 or 60). The Schott exercise treatment is also useful in this condition.

The *general nutrition* will consider above all the diet. The popular ideas which have prevailed in this respect have undergone decided changes recently. That an exclusive milk diet is a necessity in acute nephritis can not be denied, but it does not furnish the maximum of nutrition in the chronic form. In this disease nutrition is all important and, as explained under the treatment of the non-indurative form, milk alone does not accomplish this. More than that large quantities of milk flood the heart and arteries with water and thus overtax the cardiovascular apparatus. Besides the harmful dilution of the gastro-enteric secretions for this form of diet becomes very distasteful to the patient and is liable, from prolonged usage, to disturb the appetite. Croftan states an axiom when he says that "the only argument in favor of an exclusive milk diet administered for a long time is the fact that such a régime reduces intestinal putrefaction and hence removes a prolific source of renal, hepatic and cardiovascular irritation."

Starvation should not be a dietetic principle in the treatment of this disease, as nutrition is the end to be attained. The food should contain at least sufficient nourishment to maintain the nutritive equilibrium (i. e. "at least 30 calories per kilo of body weight"). To accomplish this a mixed diet is necessary as already suggested under the treatment of non-indurative nephritis. Articles of food which tend to irritate the kidney should be studiously eliminated and there should be a sufficient variety to satisfy the demands of the system.

The *restriction* of *water* is also a necessity in treatment, as it also floods the heart and arteries and thereby increases the arterial tension. In nephritis the kidney ceases to eliminate the normal amount of water, hence any effort to force them to overwork in this respect is bound to irritate them.

That water is an essential feature of food and is required by the system can not be denied, but the practice of prescribing an over-abundance of water should be discouraged.

Intestinal antisepsis is a feature of importance, as intestinal putrefaction must be avoided in order to attain perfect digestion. This is accomplished by remedies which physiologically stimulate the liver and intestine to a more normal activity. In this respect we shall receive much help from such remedies as *ox-gall* (grs. 1-3) or equal parts of *iridin* and *euonymin* (gr. 1) three or four times daily. It is also possible that this end may be attained by the more constant use of salines or in severe cases by the use of calomel. Croftan advises *sulphocarbolate* of *zinc* (gr. 1/2) or *sodium glycocholate* (gr. 1).

The treatment of the *nervous system* hardly needs attention for the importance of this is self-evident. Patients suffering with this disease must be taken away from their arduous work and all irritating influences which tend to develop neurasthenia must be eliminated. To accomplish this it is often necessary to put such patients in a rest cure or to advise a change of climate and surroundings. The specific treatment has been mentioned under the subject of neurasthenia. The *use of alcohol* must be strictly forbidden, since nothing favors the increase of arterial tension more than this.

The treatment of *acute uremia* and other complications have been outlined under other chapters and the same is true of the homeopathic remedies.

PYELITIS.

Pyelitis relates to inflammation of the pelvis of the kidney while the terms pyelonephritis and pyonephrosis relate to an inflammation of the kidney structure combined with and resulting from pyelitis.

Etiology.—Pyelitis is usually secondary to some other conditions such as urethritis, cystitis, or ureteritis. Infection of the kidney rarely takes place through the blood and only when the vital membrane of the kidney is impaired. Virulent infections may possibly affect primarily the renal structure though this organ is accustomed to eliminate all micro-organisms. Renal affections, like tuberculosis and carcinoma, may predispose to it and the same is true of decomposed urine in the renal pelvis. Stone in the kidney, other foreign bodies and irritating diuretics may favor pyelitis. Infected emboli are also factors. Floating kidney, in which the ureter becomes twisted, or obstructed, may favor a lowered renal vitality and permit an infection through the blood. Parasites must also be taken into consideration. The *micro-organisms* which are most frequently concerned in the development of pyelitis are the bacillus coli communis, the streptococcus pyogenes, the staphylococcus pyogenes aureus, the tubercle bacillus, the typhoid bacillus, the gonococcus and the bacillus proteus vulgaris.

Pathology.—In the *catarrhal* or *mild form* the pathologic change is characterized by an inflamed mucous membrane with an exudation of muco-pus and desquamated epithelium. Ecchymotic spots are occasionally present and the urine in the pelvis is turbid from the admixture of pus and pelvic epithelium. When a calculus has existed for some time ulceration is observed and inflammatory extension may develop a condition of pyelonephritis. Small abscesses or large abscess cavities may develop. A *pseudo-membranous form* may exist in the severe infections of specific fevers. Again hemorrhagic characteristics often define the pathologic condition and tuberculous ulceration is frequently present. Calcification may take place in chronic cases. In cases depending upon obstruction the dilated pelvis is filled with retained urine or pus and atrophic changes may take place in the secreting structure.

Symptoms.—The primary condition usually masks the evidence of pyelitis, an associated cystitis often being the only complaint. In a *mild catarrhal case* slight pain and tenderness in the renal region is the only manifestation aside from the possible cystitis. There may be a mild fever and the turbid urine of acid reaction reveals a few pus cells, mucus, some blood corpuscles and a slight trace of albumin. In *more severe cases,* particularly with renal colic, blood is in evidence, while pus with mucus and epithelial cells are increased. Pain is also a factor and it may be excited by jarring the body or coughing. In obstructive pyelitis, pain over the renal region is often excruciating until it is relieved by a passage of a large amount of purulent urine. The urine, in case of cystopyelitis, may be ammoniacal. Albumin is found in proportion to the amount of pus. In the *chronic variety* the pyuria is variable, epithelium being usually absent. Intermittent pyuria may occur in case of temporary occlusion of the ureter. *Septic fever* appears with the development of suppuration, a chill preceding and sweats attending the rise of temperature. Enlargement and fluctuation of the kidney may sometimes be detected in pyonephrosis.

Diagnosis.—Differential diagnosis in the case of other diseases will demand a clinical study of the case and a careful examination of the urine. *Malaria* is often mistaken for this disease but an examination of the blood should decide as to this. *Typhoid fever* likewise is often suspected and here we must rely upon the Widal test, the characteristic abdominal symptoms of typhoid together with its step-ladder temperature. *Suppurative cystitis* will be positively excluded after cystoscopic examination and ureteral catheterization. When suppurative cystitis is present there is usually a greater amount of pus in the urine, the urine is alkaline in reaction and pain is not experienced in the renal region. There is usually less albumin in suppurative cystitis than in pyelonephritis. *Paranephric abscess* is characterized by tenderness and bulging in the kidney region, while pus may not be found in the urine. A swelling may be detected in the kidney region in case of pyelonephritis but this disappears or diminishes when the pus and urine escape. *Aneurysm* may be present in painful swellings in the region of the kidney and the differentiation should be made before an operation. *Tuberculous pyelitis* is recognized when the bacilli are found in the urine. *Nephritis* is recognized when tube casts are present.

Prognosis.—The prognosis depends upon the cause, the condition of the kidney and the extent of the suppuration. Mild cases of catarrhal pyelitis appearing in the course of an infectious disease usually respond to treatment and disappear when the primary disease has been conquered. When the suppuration is sufficient to establish pyonephrosis the prognosis is not so favorable. Renal complications always add to the gravity of the case and calculous pyelitis always has a tendency to chronicity. Perforation and the discharge of pus into the peritoneal cavity, pleural sac, bronchi or intestine is invariably fatal unless surgical measures are promptly resorted to.

Treatment.—In the management of these cases we should always remove the cause if possible; this will contemplate surgical interference in many instances. *Local measures* in the form of hot applications are permissible, though they rarely give much more than temporary relief. *Diluent drinks* like flaxseed tea, barley water, buttermilk, together with a free use of alkaline mineral waters are always indicated. The *infectious cause* will demand the use of indicated remedies, local treatments and surgical help. Urotropin, uriseptin and similar urinary antiseptics may render good service. Uva ursi fluid extract (m. 5) and beta naphthol (gr. 1/10) may be given in alternation each three times daily. In the first instance in acute infections we may employ such remedies as *aconite, belladonna, cantharis, cannabis sativa, pulsatilla, rhus tox, baptisia, echinacea, terebinth, potassium citrate, buchu, chimaphila* and *pareira brava.*

In chronic pyelitis we shall find use for *berberis vulgaris, hydrastis, phytolacca, kali hydriodicum, methylene blue, hydrangea, sabal serrulata, stigmata maydis, sepia* and *silicea.*

In the *local treatments* we shall have cause to relieve urethral strictures by sounds, and to treat the prostate and urethra for the acute infections. When females are afflicted, irrigation by the ureteral catheter may be practiced. Surgery is indicated when these measures fail, when a calculus is present, or in case of abscess.

HYDRONEPHROSIS.

This is an accumulation of urinary fluid, which is not purulent, in the pelvis of the kidney because of ureteral obstruction. The pelvis and calices of the kidney may be greatly dilated on account of this and pyelitis with secondary renal inflammation and atrophy may take place.

Etiology.—Occlusion of the ureter is *congenital* or *acquired,* the former being of more frequent occurrence. The stricture is generally caused by malformation or defective development of the ureter. Rarely there is a valve like formation or an acute angled entrance of the ureter into the kidney. *Women* are occasionally affected as the result of confinement or rarely it may arise from a gonorrheal urethritis. Diseases of the ureteral walls, calculi in the ureter or pelvis of the kidney, flexion or twisting of the ureter from a movable kidney and pressure upon the ureter by tumors or pelvic adhesions are common causes. The gravid or retrodisplaced uterus, an enlarged or prolapsed spleen are rare causes, while bladder diseases which affect the ureteral orifices, prostatic enlargements and urethral strictures must also be regarded as factors.

Pathology.—The pathologic changes usually reveal a dilatation of the pelvis of the kidney. In case of prolonged pressure the kidney may become atrophic. The pressure of the fluid may also change the structure of the papillae, the tubules and the glomeruli. The mucous membrane is at first thinned and later it becomes thickened by the addition of connective tissue which may also pervade the renal parenchyma. The sac which forms may develop a nephydrotic cyst of large dimensions. The fluid usually resembles pale urine, the specific gravity is low and the reaction may be alkaline. Albumin, urea, uric acid and salts are detected. In case of previous inflammatory conditions the urine may be turbid. Hydronephrosis is usually unilateral.

Symptoms.—The symptoms depend upon the rapidity with which the renal pelvis is filled. When this develops slowly, the renal pelvis may contain a large amount of fluid before any symptoms suggest it. When it develops rapidly *pain* is often severe and is attended by a sensation of weight and dragging. A calculus or ureteral twist will cause sudden and excruciating pain. Sudden filling of the bladder when the obstruction is removed is a characteristic sign. Reflex gastric disturbances may be present and the bowel activity may be interfered with. In case of suppuration, chills, fever and sweats are added to the symptomatology while pus may be detected in the urine. Uremia is always a possible complication. Paraplegia has, in rare instances, existed as a result of pressure.

Diagnosis.—With clearly defined symptoms the diagnosis may not be difficult. The gradual development of a tumor in either flank with sudden subsidence of the same on free evacuation of urine, together with a regular diminution in the voided urine are characteristic signs. Palpation may detect a fluctuating mass beneath the floating ribs which may be mistaken for sarcoma of the kidney or of retroperitoneal glands, or, if on the left side, it may be regarded as an enlarged spleen. Tumor of the gall-bladder may be suspected when the right kidney is affected. When hydronephrosis exists in floating kidney it may be mistaken for an ovarian cyst, or in extensive dilatation of the renal pelvis, it may be mistaken for ascites. The presence of the colon anterior to the tumor should be determined. Aspiration is a positive means of diagnosis.

Prognosis.—In general it is an unfavorable disease. Compensation by the healthy kidney is favorable. Removal of the affected kidney is often necessary. A bilateral involvement is serious as uremia generally develops. Infection of the kidney is also dangerous. In rare instances spontaneous recovery may follow a rupture and discharge of the fluid, but this is rare.

Treatment.—The cause must be removed but often this is impossible. Surgical interference is generally essential. Aspiration has been performed but as the obstruction is not removed this is rarely successful. In the case of women, and less often men, ureteral catheterization has been performed.

PARANEPHRIC ABSCESS.

An abscess involving the tissue surrounding the kidney is of occasional occurrence. It exists as the result of extension of infection from the renal pelvis, injury, spinal disease, pelvic abscess, empyema, appendiceal or hepatic abscesses, and very rarely from acute infectious fevers. The tuberculous process in the kidney, carcinoma and cysts are followed by a paranephric abscess. Some cases exist without any traceable cause. A paranephric abscess may invade the suprarenal capsule.

The pus causes *pain* and *bulging* in the lumbar region, there is tenderness to touch, and in advanced cases fluctuation may be detected. The pus may burrow upward and pierce the thorax or downward and develop an apparent psoas abscess. The patient is usually prostrated, weak and emaciated and a septic temperature is in evidence. In some instances the subjective symptoms do not indicate the development of an abscess. The spine is occasionally fixed and the patient lies in bed with the limb of the affected side drawn up. The *treatment* is purely surgical.

CYSTIC KIDNEY.

Cysts of the kidney are either congenital or acquired. The congenital cysts frequently invade both kidneys and appear as a collection of cysts massed together regardless of size and shape and separated by fibrous bands or strips of atrophied renal tissue. The kidneys are, therefore, greatly enlarged and during intra-uterine life they often attain an enormous size. The cystic contents is usually a clear yellow fluid, though it may be turbid, reddish-yellow or dark brown in color; it has an acid reaction and holds in solution urinary salts, blood, cholesterin and sometimes uric acid and urea. While the true cause of these cysts is unknown they are supposed to be caused by the dilation of the renal tubules and Bowman's capsule, due to stenosis in the urinary tract or an obliteration of the tubules of the papillae. Shattuck maintains that they are due to maldevelopment of the mesonephron. It is possible for a child to live some time even when suffering with these cysts which may protrude below the ribs simulating sarcoma, hydronephrosis or splenic enlargement.

The cysts which are observed in adult life assume various forms. Small cysts are often associated with the contracted kidney as the result of dilated tubules following the increase of fibrous tissue. There may also be an aggregation of small cysts which contain clear, cloudy, thick or colloid material. These are supposed to be a late manifestation of congenital cysts or may be associated with a similar condition of the spleen or liver. Uric acid calculi in the tubules have also caused cysts. Cysts which vary in size from that of a pea to an orange may often develop in the kidney without any interference with the renal function. Solitary cystic adenoma may rarely project from the renal surface in the form of a globular tumor. On section various small cysts separated by fibrous tissue are found to compose this mass. Echinococcus cysts are occasionally found in the kidney.

The *symptoms* of congenital cyst of the kidney are often not recognized until the kidney is large enough to protrude from its natural position. As the renal structure is gradually encroached upon and supplanted by this abnormal growth the signs of chronic nephritis are apparent. *Uremia* may develop suddenly and high arterial tension with cardiac hypertrophy may be manifested. The *quantity of the urine* may be increased and the specific gravity is low, while the normal solids are also diminished. An essential feature of the urine in cystic kidney is blood. A slight or large amount of albumin may be present and Clifford Mitchell discovered the presence of acetosoluble albumin. *Palpation* will detect a large rounded or sponge-like tumor in either hypochondrium or on both sides. The *diagnosis* is made substantially by palpation. The *prognosis* is always unfavorable, and the *treatment* does not accomplish anything. Unilateral cysts have been successfully operated but the results are usually unfavorable.

NEW GROWTHS OF THE KIDNEY.

Tumors of the kidney are benign or malignant. Of the benign tumors are the *adenomas* which develop in the renal cortex in the form of small nodular masses. They may be single or multiple, rarely attain a large size, and may have a combined cystic formation. *Fibromas, angiomas* and *lipomas* are also found in the kidney. *Papillomas* may also be observed, though rarely.

Sarcomas and *carcinomas* are the usual malignant growths, which may be primary or secondary, generally the latter. *Sarcoma* is the more common form and frequently it is of congenital origin attaining a very large size in young children. The presence of striped muscular tissue in these growths points to a variety of tumor known as *rhabdomyoma* which develops during embryonic life. Alveolar sarcoma may also involve the kidney. *Carcinoma* is not of so common occurrence and when present it is of the encephaloid variety. It may rarely exist as a primary affection starting in the renal tubules. Nodular masses may develop or there may be a diffuse infiltration, the tumor sometimes attaining a large size. The abnormal tissue is liable to undergo softening, and hemorrhage is common. The renal structure is liable to destruction from these new growths and the pelvis of the kidney may be decidedly encroached upon. The pyramids are affected later than the cortex.

Symptoms.—In case of benign growths the symptoms are not marked unless they are large enough to produce pressure. When this takes place, *pain,* starting in the lumbar region, may extend along the course of the lumbo-sacral, genito-urinary and sciatic nerves. There may be only a sense of pressure or dragging in the lumbar region. *Malignant tumors* may give rise to an early sense of pain on the affected side. *Hematuria* is an early and constant sign. It may occur as hemorrhage so profuse as to cause marked exhaustion and anemia, or it may appear only as clots, which cause pain when they are expelled, or as casts of the ureter or pelvis of the kidney. The blood usually appears at irregular intervals and is often so slight that it may be overlooked. When clots accumulate in the bladder, vesical irritation is liable to take place. The urine from the healthy kidney may be found, by ureteral catheterization, to be perfectly normal. *Cancerous cachexia* is a valuable sign as the malignant tumor develops. *Progressive loss of flesh* is another very suggestive sign. *Anorexia,* nausea, vomiting and other reflex disturbances together with an increasing pallor will call attention to the organic development.

The *physical signs* are not always appreciable. As the organic growth assumes large dimensions the tumor may be palpable in either flank. This is usually possible in the adrenal sarcoma or carcinoma as both may attain a large size. To the sense of touch the tumor is hard and either smooth or lobulated. In medium sized growths the outline of the kidney may be preserved. In the large renal growths which occur in children the abdomen is greatly enlarged while the prominent veins and the abnormal pulsations are guiding symptoms. Percussion dulness is noticed over the hardened mass though, in medium sized tumors, colon tympanites is detected over the growth. Respiration usually does not displace the tumor.

Diagnosis.—The presence of a tumor in the lumbar and lower lateral region is a prominent diagnostic sign. Local pain, hematuria, loss of flesh and the cancerous cachexia are additional symptoms. The very slight mobility on respiration and the tympanitic colon in front of the tumor are also characteristic points for diagnosis. *Differential diagnosis* is often difficult. In *hepatic growths* the tumor moves freely with respiration, the area of dulness reaches a higher limit and other signs of hepatic disturbance are usually present. In a renal tumor on the right side there is generally a tympanitic area between this and the liver. In *splenic growths* the splenic notch and edge are often distinguished and there is no colon tympany over the tumor. *Pelvic growths* develop from below upward and in women a vaginal examination usually confirms its location. *Retroperitoneal sarcoma* of large size is usually more firmly fixed and centrally located.

Prognosis and Treatment.—A fatal termination is always to be expected. Removal of the kidney may be successfully accomplished when the growth is small. If metastasis has not taken place life may thereby be prolonged. The treatment otherwise is symptomatic and supportive.

DISEASES OF THE BLADDER.

CYSTITIS.

This is an inflammation of the mucous membrane of the bladder which is recognized as acute or chronic and is frequently due to infectious origin.

Etiology.—The pathogenic bacteria which excite cystitis include the gonococcus, the proteus vulgaris, the streptococci, the staphylococcus pyogenes, the bacillus tuberculosis and the bacillus coli communis. The typhoid bacillus and other infective agents may less commonly be the exciting causes. Factors which favor the development of micro-organisms such as exposure to cold, retention of urine, the presence of stone or other foreign bodies, and traumatism may be regarded as predisposing. Vesical irritation is often caused by the internal use of such drugs as cantharides, capsicum, turpentine, copaiba, etc. The use of unclean catheters and sounds is a frequent cause of cystitis, but observance of surgical cleanliness and asepsis has to a great extent overcome this danger. Adjacent inflammation, like urethritis or prostatitis, may favor the reception of a bladder infection by inflammatory extension.

Pathology.—Mild cases show scarcely any appreciable change in the mucous membrane. In other instances it is covered with mucus or muco-pus. In some cases the neck of the bladder and the prostatic urethra are alone affected. Sometimes the bladder is "ribbed" as the result of straining. In chronic cases there is a tendency toward thickening of the walls of the bladder. In the more severe inflammations, which give rise to a putrid urine, patches of false membrane are interspersed over the mucous membrane, the bladder wall also being infiltrated and undermined with pus. This has been defined as a phlegmonous or diphtheritic cystitis; as a result of this, urethral and perineal infiltration may take place. Paracystitis occurs when the inflammation invades the pelvic connective tissue.

Symptoms.—While the symptomatology varies according to the suddenness and the severity of the symptoms it is possible to recognize a difference between the acute and the chronic condition. At the same time these symptoms are so closely affiliated that a separate consideration of the two forms is not essential. *Frequency of micturition* is an early and characteristic symptom which varies according to the intensity of the inflammation. Often this takes place with such frequency that it seems almost incessant and in many instances it is attended by extremely painful tenesmus; with a subsidence of the inflammation this symptom gradually disappears. In severe cases *tenderness* over the bladder is elicited and may assume the character of actual pain on pressure. Vaginal and rectal palpation will usually reveal this tenderness and catheterization always develops it. In calculous cystitis motion and urination usually cause pain.

A *moderate fever* (100°-102° F.) is usually present in the acute forms and, should the disease partake of the diphtheritic character, the temperature may go higher and be attended by chills and sweats. The temperature of the later stages will vary according to the degree of sepsis and the absorption of putrid matter.

The *urine* may vary in each case but as soon as voided it is cloudy. In the early stage mucus mixed with pus gives it a glairy and stringy character and this increases with the severity of the condition. The reaction, when the urine is first voided, is acid or slightly alkaline, except in cases of retention with decomposition, when the alkalinity is marked. This is "due to the formation of ammonium carbonate out of the urea, the result of the operation of bacteria." The presence of muco-pus makes the passage of urine difficult. Should calculous cystitis exist, blood is liable to be a constant factor, and in the severe diphtheritic form of cystitis, gangrenous bladder tissue may also be voided. When there is extreme exfoliation of the vesical mucosa, *toxemic* manifestations are possible and a typhoid state may ensue.

Diagnosis.—*Acute prostatitis* must be differentiated from acute cystitis. In prostatitis the tumor in the rectum and the less cloudy urine will settle the doubt. There may be instances in which the two are combined. These are usually of specific origin and the history of the case will be of guiding value. *Pyelitis* independent of or associated with cystitis is not easy to differentiate. In the former, however, tenderness over the renal region may be contrasted with the tenderness over the bladder in the case of cystitis. The act of micturition is usually painless in pyelitis and the twenty-four hours' urine is increased. Also when the pus comes from the pelvis of the kidney there is some intermission in the purulent discharge. *Calculous cystitis* will be characterized by the increase of pain from motion or at the end of micturition while the urinary discharge is often suddenly interrupted and blood is observed in the urine. The use of the stone-searcher will usually confirm the diagnosis of vesical calculus.

Prognosis.—This depends decidedly upon the character of the attack. Acute cases may respond to early treatment but the chronic cases are slow to yield to any medicinal measures. The cause must invariably be removed and to accomplish this often requires the services of a surgeon, particularly when a calculus is present. An enlarged prostate will also demand surgical measures.

Treatment.—In *acute cystitis* the symptoms are relieved by early treatment. The patient should be placed in bed and copious libations of water should be prescribed. Milk diluted with seltzer is a useful agent in this respect. Some advise the associate administration of potassium bicarbonate (grs. 15-20) three or four times daily. Alkaline waters may be prescribed as needed. Diluent drinks like flaxseed, buchu, slippery-elm and couch grass are advised by E. M. Bruce. The *diet* should be bland, meats and highly seasoned foods being excluded. Asparagus, onions and rhubarb are usually objectionable. Alcoholic and malt drinks, tea and coffee should not be allowed, *Boric acid* (grs. 5-10) may be used two or three times daily for its antiseptic effect and to render the urine acid if it is alkaline. *Cystogen* (grs. 5) and *urotropin* (grs. 5) have also been employed with this object in view. These should be followed with a copious draught of water. When pain and tenesmus are severe, opium in suppository (grs. 1/4-1) may be introduced into the rectum, or the hypodermatic use of morphin (gr. 1/4) may be demanded to relieve the extreme suffering. Local treatments should be avoided in acute cystitis. An ear of boiled corn, covered with flannel and applied over the perineum often gives great relief.

The remedies which are serviceable in acute cystitis are to be administered according to their indications. *Aconite, belladonna, cantharis, stigmata maydis, chimaphila, gelsemium, pulsatilla, verbascum, triticum repens, cannabis sativa, dulcamara* and *terebinth* are of frequent service. *Mercurius corrosivus, hydrastis* and *methylene blue* are of value as the more acute symptoms begin to subside.

Treatment of *chronic cystitis* does not offer the encouraging prospect which may be entertained in the management of acute cases. The presence of the irritating urine in the bladder is always antagonistic to any treatment. The presence of mucus and pus as well as the bacteria is not overcome with ease. The irritating quality of the urine may, in a degree, be lessened by the free use of diluents and the free drinking of pure spring water always favors an improvement. The alkalinity of the urine is increased by the indiscriminate employment of alkalines. In order to make alkaline urine acid *benzoic acid* (grs. 5) four to six times daily is valuable, while *boric acid* (grs. 2-5) has also been advised. The *internal administration of drugs* to accomplish this purpose is not regarded with the favor which was entertained in the past.

The various infusions which are employed accomplish little more than diluent action. *Buchu, pareira brava, uva ursi, triticum repens, hydrangea* and various lithia compounds have been used. *Balsam of copaiba* and *sandal-wood oil* (m. 5-10) have been assiduously employed only to cause in many cases disturbance of the stomach. *Urotropin* (grs. 5), although it has its objections, is perhaps of greater service. *Boric acid* and *benzoic acid* (grs. 5-10) are of some practical use for their antiseptic effect in the chronic cases. *Resorcin* (grs. 5-10), *naphthalin* (grs. 2) and *salol* (grs. 5-10) three or four times daily have also been advised.

Vesical irrigation is our only method for applying medication directly to the bladder. This is to be performed by the Burr apparatus under the strictest aseptic precautions or by means of a soft rubber catheter which is connected with a graduated glass funnel, syphonage being accomplished by alternate elevation and depression of the funnel which contains the irrigating fluid. Sterilized water, boracic acid, normal salt solution (grs. 40-60 to the pint) or a solution of *permanganate of potash* (1:8000 or 1:4000) or a non-alcoholic *colorless hydrastis* solution may be used. The irrigation should be thoroughly performed, not more than once or twice daily, according to the demands of the case. *Vesical medication* may be attained by means of the funnel after the irrigation, the remedy being dissolved in a pint of water and allowed to run in and out of the bladder slowly. A very weak *silver nitrate* solution or *zinc sulphate* (1:5000) or saturated solution of *boracic acid* is often employed. These must be used with great caution, as a diseased bladder absorbs drugs with great rapidity. When these measures fail permanent drainage, by suprapubic, perineal, or vesicovaginal incision, may be demanded.

Some physicians use the salicylate of sodium (dram 1 to the pint of water) for the irrigation in place of the more active remedies while some substitute alum for the salicylate, a much weaker solution being used. When there is an enlarged prostate, catheterization is essential to remove the urine and the irrigation should follow this procedure.

The *internal remedies* which are useful will include aside from those mentioned under the treatment of acute cystitis, *buchu, chimaphila, equisetum, asparagin, benzoic acid, apis, sarsaparilla, berberis vulgaris* and *nitric acid.*

Stone in the Bladder.—The formation of stone in the bladder pertains to the conditions already defined under the subject of nephrolithiasis. The *symptoms* are practically those of cystitis. The occasional presence of blood in the urine, the retention of urine, the pain which occurs during urination with stoppage of the stream and the discovery of the calculus by means of a sound are the chief signs of its existence. The *treatment* is essentially surgical.

NEOPLASMS OF THE BLADDER.

Primary new growths, either benign or malignant, are of rare occurrence in the bladder. Secondary neoplasms exist more frequently, carcinoma of papillomatous formation being most common. Sarcomatous, fibromatous and cystic growths are less frequently found. The *symptoms* are generally defined by hemorrhage, frequent micturition, pain and the occasional discharge of tissue fragments. The cystoscope is an important aid to diagnosis though in advanced cases the cachexia is in evidence. When it is a secondary neoplasm the suspicion of a vesical tumor will be suggested by a knowledge of a primary growth elsewhere plus the bladder symptoms just suggested. The prognosis is unfavorable but depends upon the nature of the growth. The treatment must be surgical.

VESICAL HEMORRHAGE.

Vesical hemorrage is a symptom of malarial hematuria, leukemia, nephrolithiasis, carcinoma and stone in the bladder. It may occur independently of these conditions when there is a varicosed condition of its vessels. The hemorrhage is invariably profuse and sometimes fatal. Cystoscopic examination will aid the diagnosis but a copious hemorrhage, with the other causes excluded, will suggest this diseased condition. The prognosis is generally favorable if treatment is prompt. This is mainly local. Irrigation of the bladder with astringent solutions is indicated and the internal use of *hamamelis, secale, geranium, red-gum* and *adrenalin chlorid,* or similar remedies, are often sufficient to stop the hemorrhage.

NEUROSES OF THE BLADDER.

Vesical Irritability.—Aside from the condition of vesical paralysis which may exist as the result of organic cord diseases, we find some disturbances as the result of neuroses. Bladder irritability is an hyperesthetic state of the vesical trigone without any local pathologic condition or any discoverable organic cause back of it. It occurs among those of neurotic or hysteric tendencies and the symptoms which define these states are usually discovered upon a careful examination. It is, therefore, observed among those who are overworked, while reflex influences are frequently discovered. Thus gastro-intestinal and genital disorders are often at fault, while the hypochondriacal state and melancholia are the extreme factors which encourage it. Some of the acute infections may be the underlying etiologic factors. The irritability commonly arises from disturbances of neighboring parts as the urethra, ureter, vagina, rectum and generative organs.

Symptoms.—*Frequency of micturition,* with vesical and rectal tenesmus, is the most constant symptom and this may be attended by some pain. The passage of urine does not generally relieve the irritability or pain. A sense of weight or pressure in the pubic region is usually an additional complaint. A spasm at the neck of the bladder may occasionally overcome the voiding of urine. The character of the urine is normal but it is frequently increased in amount. This disease should not be confounded with cystitis and the prognosis is favorable.

Treatment.—The neurasthenic cause must be removed and the patient must be compelled to keep quiet. In addition to this the diet must be carefully regulated and the habits which may induce it must be corrected. The remedies which are indicated are mentioned under the subject of neurasthenia. The polyuria may be checked by the application of a belladonna plaster over the region of the kidneys together with the internal administration of *cina* tincture. *Hyoscyamus, conium, ferrum phos.,* and *piper methysticum* are useful remedies.

Enuresis.—Inability to retain the urine may arise from several causes. These may be paralytic, when the sphincteric center is involved by cord lesions, or irritative, when calculus, cystitis or parasites are present, or mechanical, when there is overdistention of the bladder or pressure exists. The cause may be spasmodic when there is an overaction of the compressor muscle with diminished vesical capacity. *Nocturnal enuresis* is a form which appears in young and neurotic children. This is often reflexly induced by phimosis, contraction of the meatus, a hooded clitoris, or ascarides. It is also a manifestation of nocturnal epilepsy or incipient cord or brain diseases. Diabetes must be looked for in enuresis with polyuria.

The *treatment* depends upon the cause, which must always be removed. The general health, the surrounding influences and the habits always need careful attention. Hygienic care is essential and the pride should always be appealed to while the habit may sometimes be broken by proper discipline. Constipation should always be corrected. Sleeping on a hard mattress or with a towel knotted over the lumbar region is often effective.

There are many homeopathic remedies which may be used successfully in these cases depending of course upon the cause and the symptomatology. *Benzoic acid* in the third decimal potency has served me better than any other remedy. *Pulsatilla* is particularly useful in nocturnal enuresis or when there is a tendency to dribbling of the urine during the daytime. *Equisetum* also pertains to nocturnal enuresis but it is more useful in older children. *Apis* may be used when there is burning and soreness following the discharge of urine and the stinging pain is also recognized. *Cina* is naturally thought of when worms are at fault, while *arsenicum, ignatia, nux vomica* and similar remedies may be employed for gastro-intestinal irritations. *Gelsemium* refers to a bladder paralysis with nervous causes underlying. *Belladonna* applies to that class of patients who are restless at night and start in their sleep at any unusual noise. *Valerianate of ammonia, agaricin, picric acid, asafetida, moschus* and many other remedies may be indicated. Hypnotics may be required in severe and protracted cases. *Rhus aromatica* has often been found very serviceable in this condition.

Retention of Urine.—Nervous retention occurs in hysteria and from any excessive excitement in neurotic patients. Sometimes the urine can not be voided in the presence of others. It occurs after serious nervous shock when the bladder is temporarily unable to contract upon its contents with sufficient force to relieve it. When the urine remains a long time in the bladder decomposition takes place and cystitis may follow.

The *treatment* will include the restoration of the normal nervous state, the relief of any local excitement and the employment of indicated remedies.

DISEASES OF THE NERVOUS SYSTEM.

DISEASES OF THE PERIPHERAL NERVES.

NEURITIS.

The peripheral nerves are subject to inflammatory invasion which is termed neuritis. This may affect the nerve-sheath or the septa between the fasciculi. When the inflammation affects the nerve-fibres chiefly, and takes on the degenerative process, it is termed *parenchymatous neuritis.* When the inflammation extends from adjoining structures the sheath alone is affected and it is termed *perineuritis.* Neuritis is either *focal* or *diffuse* and when several nerves are involved it is called *multiple neuritis.*

Focal Neuritis.—Etiology.—Compression or contusion are frequent etiologic factors. Thus fractures, dislocations, muscular strain and wounds may be the inciting causes. Exposure to cold and extension of inflammation by contiguity also cause this disease. Micro-organisms and autotoxemias have an etiologic influence.

Pathology.—The inflamed nerve is usually red and swollen; its sheath is also involved and shows round-cell infiltration. The inflammatory and degenerative process is generally confined to the area of the lesion, though in case of a severe contusion it may take the form of a descending degeneration, while in case of infected wounds the neuritis may extend upward. Distinct tumors are sometimes observed on the affected nerve.

Symptoms.—The *onset* is usually abrupt and pain is then an early symptom. This is confined to the site of the inflammation, tenderness being detected on pressure over the nerve-trunk and its distribution. Pain is invariably increased by muscular action. Paresthesia, hyperesthesia and often diminished sensitiveness may exist over the cutaneous area involved. After degeneration begins skin anesthesia is present over the affected part. These sensory disturbances are uniform and constant. *Impairment of motion* is observed in the muscles supplied by the affected nerve. This may amount to a complete paralysis. Contractures and fibrillary twitching are often detected and the reaction of degeneration will be found as the trophic loss becomes extreme. Atrophy and other trophic changes will be evidenced as the disease progresses. When both motor and sensory nerves are involved the symptoms will represent a combined motor and sensory perversion. Constitutional symptoms are rarely present.

Diagnosis and Prognosis.—The *diagnosis* will be established by a careful study of the foregoing symptoms. The *prognosis* is usually favorable if prompt and energetic treatment is applied. The removal of the irritating cause usually cures the disease. A slight neuritis responds to rest and the indicated remedy. Traumatic cases improve more promptly than those caused by extending inflammations. An upward extension of the neuritis is unfavorable. A degenerative lesion is slow to heal and always gives an unfavorable outlook.

Treatment.—Rest, warm applications, removal of the inciting cause, and immobilization of the involved muscles are demanded in the early treatment. Later, massage and electricity should be employed. Anodynes are necessary when pain is severe. *Aconite, belladonna, hypericum, arnica, calendula, bryonia, gelsemium, rhododendron, rhus tox* and similar remedies are required. Later *agaricin, zinc phos., mercurius, aurum mur., arsenicum iodid* and other iodids are required.

MULTIPLE NEURITIS.

Multiple neuritis, polyneuritis, beri-beri or kakké refers to the inflammatory invasion of several nerves, the affection often appearing simultaneously in various parts of the body.

Etiology.—The cause of multiple neuritis is traced to *toxic substances* like lead, alcohol, arsenic, mercury, etc., which, introduced into the system, exert their poisonous force upon the nerves; or it may be due to *autotoxemias* which frequently attend diabetes, nephritis and intestinal disturbances. The *infections,* like typhoid, the exanthemata, erysipelas, influenza, rheumatism, diphtheria, etc., are also direct causes, while syphilis and tuberculosis particularly predispose. The *cachexias* frequently incite it, in old age it attends arteriosclerosis and certain occupations may favor it.

Pathology.—The pathology is observed in the structure of the nerve and extends upward, the root rarely being affected. In the mild form the degeneration simulates that which is secondary to traumatism, but in the more severe form interstitial inflammation and degeneration are in evidence and are usually diffuse. When the interstitial inflammation is pronounced slight hemorrhage, exudation and leukocytes are observed about the vessel walls. Atrophy of the muscles involved and occasionally interstitial myositis are recognized. When the inflammatory process continues to any extreme the nerve fibres degenerate, a connective tissue supplanting them. The *anterior horn cells* may infrequently show signs of the degenerative process and there may be a similar though slight involvement in the posterior and lateral columns. The cells of the *cerebral cortex* may display atrophic degeneration, particularly in the case of alcoholic poisoning. As a rule, however, the cerebral and spinal cord changes are not marked.

Symptoms.—Weakness of the lower extremities and sometimes the upper exists for several weeks before the patient begins to complain of numbness, a burning sensation in the hands and feet and other paresthesiae. There is usually some evidence of mental confusion and occasionally insomnia. A *sudden onset* may characterize the symptomatology, the patient often suffering with such pain in the legs and feet that it is necessary to go to bed for relief. A *slight fever* (102°-104° F.) exists for a time, but this is not long lasting. The *pains* and weakness in the limbs constantly increase, compelling the patient to take to his bed. *Edema* and redness of the skin, together with other signs of trophic and vasomotor disturbance, are frequently early symptoms. By the *end of two weeks* the anterior tibials are completely paralyzed, while the extensors of the hands are also considerably affected. Other muscles are soon involved, the pain becomes more distressing and atrophy begins. The motor cranial nerves are occasionally affected. In at least two weeks more a *complete paraplegia* exists; *foot drop* is marked, *wrist drop* is more or less in evidence, while muscular atrophy is more advanced and the feet are usually swollen. The knee and elbow reflexes are always lost; this is sometimes true of the skin reflexes. The temperature and pain sense are impaired and retarded, tactile anesthesia is recognized and hyperalgesia is often pronounced. Anesthetic patches, perversion of the muscular and articular senses, always in the pseudo-tabetic type, are other characteristic symptoms. The *pain* continues constantly and may respond to no medication.

The *nerve changes* are manifested in the loss of their irritability; the muscles show a partial and often complete reaction of degeneration and an early loss of faradic, and lessening of galvanic, irritability. The *pneumogastric nerve* is frequently affected, giving rise to cardiac excitement and respiratory disturbances. The *sphincters* are involved only temporarily, if at all. *Mental disturbances,* characterized by a low muttering delirium or confusional insanity of short duration, are infrequently observed. The *active features* of the disease are limited to two or three weeks when it usually assumes a chronic stage with occasional acute exacerbations which may be as severe as the primary attack. The typical *alcoholic cases* suffer from extreme exhaustion and are liable to die from intercurrent disease. In *diphtheritic neuritis* throat symptoms are prominent, the muscles of the throat and also the eye being temporarily paralyzed while the extremities are only slightly affected.

The *motor symptoms* of a typical multiple neuritis are characterized by a quadruplegia; in the alcoholic form the foot drop is as distinctive as the wrist drop is characteristic of lead poisoning. It is a peripheral paralysis in which the feet, legs, hands and forearms are chiefly affected. The muscles become weak, flabby and atrophic. The *early loss* of faradic and galvanic reaction is a characteristic differentiation from similar diseases. Galvanic reaction being lost last, this irritability returns first in case of improvement. *Contractures* may appear if the paralysis lasts long during the chronic stage.

Acute Pernicious Multiple Neuritis.—Dana describes a form of pernicious neuritis which "develops suddenly, develops rapidly and causes death in a few days." In addition to the ordinary neurotic symptoms the cardiac and respiratory centers are involved, this being the cause of death. The causative factor is supposed to be some form of sepsis and the pathology is characterized by interstitial and hemorrhagic changes. The anterior horn cells are also involved.

NEUROMAS.

Neuromas are classified as nerve hyperplasia, and also as true and false neuromas. *Hyperplasia* or hypertrophy of nerve trunks is uncommon; the increase in size is usually due to an increase of the interstitial connective tissue. More rarely there is increase in the number of nerve fibres with thickening of the myelin sheath. *True neuromas* are rare and are usually found only on the spinal nerves. There may be only a neurilemma and axis cylinder increase or a medullary fibre increase. They may be single or multiple, the latter being neuro-fibrous in structure. The neuro-fibromas often form growths known as fibroma molluscum. Plexiform neuro-fibromas are nerve fibres united in a mass by hyperplastic connective tissue. The true neuromas are usually small and few in number.

False neuromas are such tumors as fibroma, myxoma, glioma, sarcoma, carcinoma and syphiloma. Malignant neuromas are very rare. *Tubercula dolorosa* are small false neuromas located on the ends of sensory nerves. *Plexiform neuromas* are beaded and tortuous interlacing neural cords which are of congenital origin.

Etiology.—A neuropathic predisposition accounts for most of the true and the plexiform neuromas, traumatism is the cause of the fibro-neuromas, while diathesis must be considered in the tuberculous and carcinomatous growths.

Symptoms.—Exacerbating *pain* and slight tenderness are the most common indications of neuromas, though they often exist without any distinctive signs. Paresthesia, anesthesia, paralysis, and reflex spasm are commonly observed, while compression symptoms often arise from the development of malignant neuromas.

Diagnosis and Treatment.—The diagnosis of neuroma can be positively made only by palpation, and is really made by exclusion. The treatment is purely surgical, as internal medication gives no relief.

NEURALGIA.

Neuralgia is characterized by pain which is distributed or reflected along the course of a nerve or a plexus of nerves. It is an expression of some local or systemic disturbance or a combination of both. Neuralgias are classified according to location or cause. Thus trifacial, cervico-occipital, cervico-brachial, intercostal, sacral, coccygeal, etc., are regional varieties, while pleurodynia, angina pectoris, cardialgia, gastralgia, gastrodynia, nephralgia or ovaralgia, etc., are terms descriptive of the viscera involved. Those depending upon specific or general causes are defined as epileptiform, reflex, sympathetic, traumatic, occupation, herpetic, anemic, rheumatic, malarial, etc. The differentiation between neuralgia and neuritis is difficult to establish. While neuralgia is one of the symptoms of neuritis, or if it exists from other causes for any length of time may develop a neuritis, it is still only a symptom of some direct or indirect disturbance of the nerve or nerves affected.

Etiology.—This naturally embraces such predispositions as *age,* inasmuch as it rarely appears until after puberty, and if it exists in old age it is of organic origin and incurable. *Women* seem to suffer from it more than men and *heredity* must always be taken into account in the history of every case. *Hysteria* or *epilepsy* may be underlying factors, while neuroses and psycho-neuroses are commonly predisposing influences. The *general health* has more to do with the development of neuralgia than is often apparent. Mental and physical exhaustion, no matter from what cause, insomnia and particularly anemia, are so often at fault that the physician should always take these factors into consideration. *Occupation* which predisposes to nerve strain, general exhaustion or infection is a factor in the etiology. *Reflex disorders,* particularly of the gastro-intestinal or genito-urinary tracts, are frequently the underlying causes and should always be looked for. Under *inciting causes* may be included exposure to cold, local irritation, neuritis, toxic influences, diabetes, lead and alcohol being pre-eminent; autointoxication, malaria and other acute infections also play a part in the cause of neuralgia. We must also take into consideration that class of diseases of the mouth, throat, nose and orbit which may cause local irritation or pressure along the course of the fifth nerve.

Symptoms.—The *pain* of neuralgia is excruciating and paroxysmal, the seizure being usually characterized by its sudden onset. It has also a unilateral location and is generally confined to the distribution of a part or the whole of a single nerve. It has been more minutely described as stabbing, tearing and darting. It is deepseated, but is characterized by extreme superficial sensitiveness. Deep pressure usually gives relief. Motion of the supplied muscles, the slightest touch or breath of air usually excites the paroxysm. *Tenderness* is a marked feature of the symptomatology and is most clearly manifested when the nerve is superficial, overlies bone or is enclosed by fascia. *Radiation* of the pain to branches of the nerve or to related nerves is quite common and its migratory character is recognized, particularly when there is a systemic cause. *Spasmodic contraction* of the associated muscles is also a feature of the pain storms. The *vasomotor symptoms* are characterized by flushing or blanching of the affected part or an increase in the secretions. *Trophic disturbances* are represented by herpetic and urticarial eruptions and the more permanent perversions of this kind are characterized by color changes, loss of hair, pigmentary changes and possibly ulceration.

Trifacial Neuralgia.—This is one of the most typical neuralgias with which we have to deal. It is also the most common form, inasmuch as the extensive distribution of this nerve renders it extremely liable to peripheral irritation and injury. This is explained by the fact that it is exposed in the nose, nasopharynx, face and bony channels, to much irritation. By its intimate relation with other nerves it is also subject to reflex irritation.

When the disturbance is confined to the *ophthalmic branch* the pain will be distributed to the nose, eyes and forehead; the eyeball is tender and may be the seat of the pain. When the *superior maxillary branch* is implicated the pain is localized over the cheek and spreads out toward the wing of the nose, involving also the hard palate. When the *inferior maxillary division* is affected the pain is located more in the lower jaw, the tongue and the corresponding portion of the face.

The character of the pain in trifacial neuralgia has been defined as "excruciating torture" and, in the majority of instances, this is not overdrawn. The vasomotor disturbances which attend are lachrymation, salivation and nasal discharge. Edema is occasionally observed over the part and hyperalgesia is intense.

Treatment.—The treatment of this form of neuralgia will apply to the majority of neuralgias. The location and relief of the irritating cause is the first therapeutic object. Careful palpation for painful points will aid us decidedly in localizing the seat of trouble. Besides this a thorough examination of the eye, the nasal fossae, the antra, the nasopharynx, the mouth and the jaws are essential. A dentist must also carefully examine the teeth. The systemic condition must be taken into consideration, for many cases are due to infections like influenza, malaria, autotoxemia, etc. Anemia, cachexia and autointoxications are so frequently the cause that great stress should be placed upon these factors. Eyestrain, which so commonly affects the ophthalmic division, must always be looked for, compression of the nerve in the dental canal must be investigated and diseases of the teeth and jaw must be thought of in the treatment. Nerve impingement is often a factor which must be corrected before a cure is attained.

Specific treatment for the relief of pain is usually sought as no patient will endure this neuralgia without temporary relief of some kind. *Local applications* of all kinds are usually resorted to without much relief. Sometimes, however, heat or counter-irritation or even ice palliates for a time, if they are applied to the painful point. Soothing liniments such as the standard belladonna liniment, camphorated oil, etc., sometimes gives relief for a time, but this is not long lasting. Equal parts of chloral hydrate and camphor gum make a soothing lotion which gives much temporary relief. *Anodynes,* however, will always be demanded and they are often our only hope until the cause is removed or a clearly indicated remedy is found. *Morphin* is absolutely demanded to subdue the excruciating pain, and this is best administered hypodermatically (grs. 1/8-1/4). *Cocain,* bromids and many hypnotics are usually resorted to, but they do not give the relief that morphin does. *Strychnia,* in increasing doses hypodermatically administered, has been commended by some, but its universal application is not successful. *Ergotin, atropin, hyoscyamin* and *digitalin* have also been recommended, but no uniform results have been obtained by their use. Operation, either the section of the involved nerve branch, excision of a portion of the nerve or removal of the Gasserian ganglion, is also advised, but it should be a last resort. Moyer has obtained good results from castor-oil (drams 1-2) three or four times daily.

One of the best homeopathic remedies for the relief of neuralgia is *physostigma.* This refers to the pains which affect the ocular muscles, or the severe pain is located in the region of the eye. It also has a direct relation to symptoms which refer to nasopharyngeal disorders, and spinal irritation is to a certain extent covered by this remedy. Physostigma may be compared to *conium, curare* and *gelsemium.* The next remedy which I prefer when nerve irritation is well established is *agaricin.* With this we may compare *moschus, asafetida, valerian, sumbul* and *ignatia.* When there is evidence of trophic disorder *ferrum phos., zinc phos.,* and *zincum pic.,* are frequently indicated. *Coffea, hyoscyamus, stramonium, colocynth, camphor, glonoin, cuprum* and *cicuta virosa* will answer in many cases of long standing. *Atropin* may be cautiously employed, in increasing doses, when there is a general spasticity of the affected nerve. *Mezereum* is a useful remedy when there are associate diseases of the skin. With this we may compare *mercurius, rhus tox* and *arsenicum.*

Neuralgia of the Neck and Trunk.—*Cervico-occipital neuralgia* or neck pains may occur from many causes. It is a feature of migraine, hysteria, spinal irritation, eyestrain, neurasthenia and many other diseased conditions, but when not due to some underlying organic cause the prognosis is fair and it usually responds to symptomatic treatment.

Intercostal neuralgia, or side pain, is a common form of neuralgia. It comes often from muscular strain or exposure and it is also an attending symptom of other diseases like anemia, neurasthenia, hysteria, malaria, heart disease, and also the infections. The pain is sharp, lancinating and radiating; all movements of the chest intensify it. When this neuralgia persists for a long time we may suspect a disease of the spinal cord, neoplasms, aneurysm or vertebral disease. *Mastodynia* is a form of intercostal neuralgia involving the anterior and lateral branches of the three or four upper dorsal nerves. It is usually caused by mammary tumors or it may be an essential neuralgia.

Neuralgia of the Extremities.—*Cervico-brachial neuralgia* is not very common and usually arises from the ordinary causes of neuralgia, gout and rheumatism especially predisposing. Muscular strain, anemia, and reflex irritations are occasional factors in its existence. It is likewise an occasional symptom of a cord lesion and may be a residual neuritis. The lower four cervical nerves are most commonly affected and when it is bilateral a cord lesion may be feared. The *symptoms* are characterized by aching pains in the neck, shoulder, axilla and along the nerve affected. Use of the arm, pressure or exposure excite the suffering while exacerbations of pain generally occur at night. *Painful points* are felt in the axilla, at the lower end of the scapula, over the deltoid and other superficial points of the nerves involved, as a general thing all the nerves of the plexus being affected. *Digital neuralgia* is sometimes a feature of this form of neuralgia, the pain often being located in a single finger as the result of injury or neuritis.

Femoral or *crural neuralgia* is a rare form which attacks the anterior surface of the thigh, the knee-joint and the inner surface of the leg and foot.

Obturator neuralgia is located on the inner side of the thigh and down to and including the knee-joint. It is common in women suffering with ovarian disease. *Sciatica* is one of the most common forms of neuralgia, men in middle life being most frequently affected. The gouty and arthritic diathesis, and occupations which predispose to exposure and strain are the usual causes. A neurotic tendency also has its influence. Constipation, pelvic disorders and inflammation of the hip-joint are also liable to develop it. The *symptoms* generally appear suddenly, a severe stabbing pain being located along the sciatic nerve and extending well up into the lumbar region. Motion always increases the pain and the patient holds himself in a cramped position to obviate this. A spinal scoliosis, the convexity of the vertebral curve being directed toward the diseased side, is sometimes recognized. The pain constantly increases with unbearable nocturnal aggravations. Numbness and other paresthetic disturbances are occasionally in evidence.

Tender points are found all along the nerve, the sciatic notch, the middle of the hip, behind the knee, below the head of the fibula and the back of the foot being the most common tender points. Pressure over the back of the knee when the leg is extended at more than a right angle will start a pain up the back of the thigh. With the patient lying upon the back and the leg extended, a sharp pain is felt in the sciatic notch when the limb is elevated until it reaches an acute angle. These last two symptoms are diagnostic. Muscular wasting may occur in long-lasting cases. The disease generally has a long duration and is subject to recurrence on the slightest provocation, the sacral plexus and the spinal cord occasionally being involved. It must be distinguished from hip-joint disease, organic disease of the cauda equina or cord, muscular disturbances and pain caused by the pressure of tumors.

The *treatment* is similar to that of trifacial neuralgia, though rest should be enforced. It is well to immobilize the limb with the patient constantly in bed. The oil of gaultheria is often a soothing lotion externally and turpentine in full doses (m. 5-15) is sometimes of great relief internally. Nerve stretching has been abandoned and little relief is obtained by dissecting the nerve sheath.

Plantar neuralgia is experienced when the nerve disturbance is limited to the plantar nerves, paresthesia and marked anesthesia of the parts being noticed. *Erythromelalgia,* or red neuralgia, is a neuralgia affecting the feet and characterized by burning pains and congestion of the parts. *Morton's neuralgia* is a neuralgia affecting the metatarso-phalangeal joint of the third and fourth toes. It is due to a slight luxation with constant pressure on a digital branch of the external plantar nerve. *Tarsalgia* is a neuralgia due to incipient flattening of the foot and stretching of the plantar ligaments. It is noticed frequently in policemen. *Coccygodynia* is a neuralgia affecting the lower posterior branches of the sacral nerves. It occurs reflexly from pelvic disorders and it may be a result of spinal irritation. It interferes with walking, the parts are tender and pain is experienced at stool. *Visceral neuralgias* may affect various viscera, the gastro-intestinal tract being most subject to the disturbance. The treatment of all these neuralgias should be symptomatic and palliative, as has been described under the treatment of trifacial neuralgia.

DISEASES OF THE CRANIAL NERVES.

The Olfactory Nerve.—When any disease of the olfactory nerve interferes with the sense of smell *anosmia* exists. A partial loss may arise from lesions of the peripheral ending of the nerve in the nasal mucosa. Injury to the nerve-trunk or bulb, meningitis, and bone-disease may cause a partial or complete loss of the olfactory sense. Central lesions which affect the bulb or tract usually give unilateral anosmia which is generally permanent. Neuroses or hysteria may also cause a temporary loss of the olfactory sense. *Hyperosmia*, or abnormal acuteness of the sense of smell, is of rare occurrence and is often a feature of hysteria or insanity. *Parosmia* is a perversion of the olfactory sense. It may be the result of hysteria or due to local disturbance. The *treatment* should consider only the removal of the cause.

The Retina.—Hemorrhage into the retina may be single or multiple, monocular or binocular. It may be venous or arterial and is often an expression of a general vascular change. Nephritis, anemia, leukocythemia, malaria, purpura and parturition are held to be causes. Vision is partially or completely lost as the result of this hemorrhage and may occur suddenly or gradually.

Retinitis.—This assumes several forms. (1) *Albuminuric retinitis* is associated with nephritis and is a feature of the general fibro-vascular change which takes place in that disease. The loss of vision and the advent of albuminuria usually occur together. Chronic interstitial nephritis is most frequently attended by this. The retinal changes are either hemorrhagic or degenerative. (2) *Syphilitic retinitis* occurs in the later stages of acquired syphilis. (3) *Pigmentary retinitis* is a chronic condition occurring in those afflicted with inherited syphilis or suffering from a low state of vitality.

Other retinal affections may also occur. *Toxic amblyopia* is usually caused by the excessive use of alcohol and tobacco or it may result from other poisons. The vision fails gradually, absolute blindness rarely resulting. Recently many cases due to drinking wood-alcohol have been observed. *Hemeralopia* or day-blindness is due to some retinal affection or is a functional perversion. *Nyctalopia* or night-blindness is a diminution of vision in a darkened room or after nightfall, a strong light being always needed for normal vision. It is often associated with syphilitic retinitis.

The Optic Nerve.—Under this head three pathologic conditions exist. (1) *Perineuritis* occurs in meningitis and in some of the fevers. (2) *Choked disk* occurs in intracranial tumors, nephritis and syphilis. (3) *Neuritis* is observed in syphilis, nephritis, intracranial tumors and more rarely, in anemia and lead poisoning. (3) *Optic atrophy* is a feature of tabes and may arise from certain toxemias or diabetes; when hereditary it is known as Leber's disease.

The Optic Tract.—The lesions of the tract may or may not show retinal changes, though these usually exist in prolonged cases. Lesions of the chiasm, affecting the decussating fibers, cause blindness of the nasal halves of the retina and temporal hemianopsia. Tumors at the cerebral base, acromegaly, tuberculous basal meningitis and hydrocephalus may be the causes. Complete lesions of either optic tract cause homonymous hemianopsia; if this is incomplete the visual field is irregularly disturbed. Other structures are often simultaneously affected. Hemorrhage, tumors, cerebral softening and basilar meningitis are causes. Lesions anterior to the anterior corpora quadrigemina usually cause some destruction in other cranial nerves, giving rise to ocular palsies, special sense disturbance, anesthesia or neuralgia. In lesions of the optic thalamus or the internal capsule, hemiplegia and hemianesthesia are important symptoms. Lesions posterior to the anterior corpora quadrigemina cause hemianopsia without disturbance of the pupillary reflex. These lesions are cortical and subcortical and are either irritative or paralytic. Those of irritative character give rise to hallucinations of vision, while paralytic lesions cause hemianopsia. Bilateral lesions may or may not cause total blindness in one eye. Cortical lesions of the tract involve the occipital lobe; if the cuneus and calcarine fissure are destroyed, total blindness follows. Mind-blindness occurs when the convex portion of the occipital convolutions are affected. Hemianopsia is a temporary condition when the result of uremia, apoplexy, migraine and certain intoxications. It is permanent when the visual tract is affected by a destructive lesion.

Diseases of the Motor Nerves of the Eyeball.—The *motor oculi* is subject to *spasm* or *paralysis*. In the *former* the internal rectus and levator palpebrae are most frequently affected. It is incident to meningitis, hypermetropia and hysteria; a clonic and bilateral spasm is noticed in nystagmus. When the levator palpebrae is chiefly affected the eye can not be closed; stimulation of the nucleus or the nerve may cause myosis as in tabes; sympathetic paralysis causes a similar effect. *Paralysis* of the nerve may take place as the result of inflammatory exudate, tumors or disease involving the nucleus. Relapsing palsy occurs most frequently in syphilitic conditions, one muscle being paralyzed after the other has partially recovered. Recurring palsy is a rare form resembling migraine which occurs in hysterical women. General paralysis of the extra-ocular muscles gives rise to ptosis, divergent strabismus, diplopia, and contracted pupil with loss of light reflex and accommodation.

Intra-ocular Paralysis.—Ciliary muscle paralysis or *cycloplegia* perverts the power of accommodation, causing near-sight to be blurred and indistinct, while far-sight is good. A bilateral cycloplegia sometimes occurs in the course of diphtheria or tabes as the result of a nuclear lesion. *Iridoplegia* is the paralysis of the fibres of the third nerve which are distributed to the iris, resulting in dilatation and immobility of the pupil. *Myosis* is the direct opposite of the above condition resulting from sympathetic paralysis when the motor fibers of the iris are paralyzed. The pupillary reflex is lost in tabes, general paresis and occasionally in peripheral disease of the third nerve. Syphilis is also a factor. The skin-reflex iridoplegia is characterized by reflex dilatation of the pupil. The accommodation iridoplegia is observed in general paresis.

Fourth nerve paralysis is manifested by downward and inward movement of the eye. In consequence of this there will be a diplopia while the head is usually tipped forward and to the sound side. When this nerve alone is affected there must be a lesion of the nucleus. The *sixth nerve* supplies the external rectus muscle and when paralyzed convergent strabismus exists because the eye is not rotated outward and thus diplopia follows. This nerve throughout its long course is liable to injury or pressure from tumors; it is also subject to the effects of syphilis and meningitis. In case of a nuclear lesion the external rectus of the same side and the internal rectus of the opposite side are paralyzed and thus a conjugate deviation follows, as there is communication between the sixth and the third nerves.

The *general symptomatology* of paralysis of these eye-muscles embraces the limitation of movement which depends upon the extent of the palsy, strabismus which refers to the perverted axis of the eye, secondary deviation caused by the over action of the unaffected muscles, erroneous projection, double vision and homonymous or simple diplopia. These paralyses may be partial or complete, may be of sudden or gradual onset and the sequelae will depend upon the cause and the extent of ocular impairment. These cases should be sent to a competent oculist for treatment.

Diseases of the Fifth Nerve.—Lesions of this nerve give rise to either sensory, motor or gustatory disturbance as the nerve is both sensory and motor in its function. Originating in two nuclei in the floor of the fourth ventricle and developing several ganglia in its course, it is subject to various pathologic disturbances. Hemorrhage into the pons, tumors, softening and sclerosis in this region, and basal traumatism may give rise to disease of this nerve. Peripheral neuritis and injuries to the skull or brain may also be inciting factors.

The *symptoms* which follow when the *sensory part is affected* are characterized by the most excruciating pains which have been discussed under trifacial neuralgia. There is also local tenderness, hyperesthesia and later anesthesia. Such parts as the mucosa of the nose, mouth, lips and tongue reflect the local sensory disturbance. Muscular movements may be retarded by this sensory impairment. The secretions are first increased, then lessened, while the loss of taste and smell may be added to the trophic perversions of the parts receiving its sensory branches. When the *motor branch* is implicated there will be partial or complete loss of movement affecting, of course, the muscles of mastication. Spasms of the muscles, either tonic or clonic, may also take place when the fifth nerve is affected. Tonic spasm is observed in trismus or lockjaw and may have tetany, hysteria, caries of the teeth or irritative central or peripheral lesions as the underlying cause. Clonic spasms may appear in paralysis agitans, general convulsions or climacteric disorders. Gustatory disturbances are occasionally observed when the nucleus of the fifth nerve is affected. A loss of the sense of taste is more liable to occur in association with middle ear disease, hysteria and insanity. Irritative lesions may cause increased sensitiveness.

The *treatment* of fifth nerve disturbances will look to the primary cause which must be removed in each instance. Analgesics and opiates are often required for temporary relief, and internal medication embracing iodids, mercury, the salicylates and other indicated remedies is invariably required.

Diseases of the Seventh or Facial Nerve.—*Spasm* of the facial nerve may arise from organic or idiopathic causes and the nerve may be partially or entirely affected. Psychic disturbance is a common cause while peripheral or reflex irritation are factors. The lesion may appear in any part of the motor tract of the nerve; thus the cortex, the facial nucleus or the nerve itself may be implicated. A neuropathic heredity, emotional disturbance, diseases of remote organs and organic lesions may reflexly or directly produce the spasm. In some it is a pure case of mimicry.

The *symptoms* are particularly characterized by the spasm which is usually a sudden clonic convulsion of the muscles of one side of the face, closure of the eyelid and retraction of the angle of the mouth being most frequently observed. The spasms may occur in groups frequently repeated, may occur at irregular periods or a single spasm may take place. Rarely the spasm is tonic in character and lasts for several seconds or minutes. Sometimes it is associated with a general tonic convulsion. Pain is not a feature of facial spasm, but a sensitive point may be determined over the supra-orbital foramen. Tinnitus aurium sometimes exists and more rarely taste is disturbed. Facial spasm may be an associate movement. It may be confounded with chorea. A cure is not obtained in the organic cases of long standing, but when the source is peripheral it may be accomplished. *Treatment* should rely upon the use of electricity and internal remedies. Operative interference is not usually successful. *Facial paralysis* may be of central, nuclear, meningeal or peripheral origin. When it is central it is due to hemorrhage, softening, inflammation or tumor of the brain. Palsy of nuclear origin is extremely rare and when there is a meningeal cause other cranial nerves are usually affected.

Peripheral facial palsy (*Bell's palsy*) is the most common form of facial paralysis. Infection and exposure are the most prominent factors in its occurrence, and it is inclined to appear in the early and middle part of life. Syphilis is rarely a factor and heredity, other than a neuropathic tendency, is of slight influence. Non-typical cases may arise from injury, fracture of the petrous portion of the temporal bone or ear disease. Some cases may follow prolonged labor and the use of forceps.and the disease may rarely be congenital.

The *symptoms of Bell's palsy* are of sudden appearance and reach their height within a few hours or, at least, in a few days, without any marked premonitory signs. The patient observes that the eye of the affected side can not be perfectly closed, that the corner of the mouth is drawn toward the unaffected side when used, that mastication is not properly performed, the lips can not be puckered and speech is imperfect. The facial appearance is characterized by the lack of wrinkles on the affected side of the face, the lowered angle of the mouth and the unnatural appearance of the affected eye, all of which symptoms are aggravated when the patient attempts to laugh. When the patient is told to shut the eyes firmly the affected eye is not closed. In the majority of cases the nostril of the affected side does not expand on forced inspiration. The loss of taste may ensue when the seventh nerve is involved between the geniculate ganglion and the point where the chorda tympani is given off. When the sense of taste is not involved we may know that the lesion is peripheral.

Atrophic changes may affect the muscles involved if the disease continues for any length of time. During the first few days there is an increase of both faradic and galvanic irritability. The faradic irritability is soon lost and the galvanic disappears a little later. The electrical reaction usually returns in a few weeks or sooner if there is prompt treatment.

The prognosis of peripheral palsy (Bell's) is good and most of the cases recover if early treatment is undertaken. In central palsies there is not so much hope as the lesion remains. The duration of the peripheral palsies is from three to five months. Milder cases which receive prompt attention get well sooner. A syphilitic etiology is always unfavorable.

Treatment.—The *early* treatment should include the enforcement of absolute quiet in bed during the acute stages, the use of a mild cathartic and hypnotics if the patient is inclined to be excited and worried. Remedies like ***aconite, belladonna, hyoscyamus, arnica, hypericum*** and ***cimicifuga*** are indicated early. Usually no other medication is demanded. Some advocate the use of sodium salicylate (grs. 10-20) two or three times daily. This or any remedy increasing elimination may be useful. Electricity should be carefully employed after the first week and daily treatment should be given after this for five or six minutes at each sitting. Galvanism usually answers best at first, but faradism should be substituted for this as soon as that current will cause contraction. After a month's time the treatments may be less frequent. Massage may follow the use of electricity at the end of two months at least. The remedies which may be used in the later stages are ***strychnia, arsenicum iodid, physostigma, gelsemium, cimicifuga, zincum picricum, phosphorus, ferrum phos.,*** and ***argentum nitricum.***

Diseases of the Eighth Nerve.—*Irritation of the auditory* nerve gives rise to auditory hyperesthesia, increased acuteness of hearing (hyperacusis) and tinnitus. Continued irritation may result in complete loss of hearing. Hyperesthesia may occur under various conditions, hysteria and neurasthenia being common causes. Meningitis, acute mania and drug poisoning may give rise to it. Hyperacusis is rare, though migraine and pain may make a patient more sensitive to sudden noises. Paralysis of the stapedius muscle gives greater intensity to low notes. Any disturbance of the auditory mechanism may produce tinnitus. Foreign bodies or cerumen in the external meatus, inflammation of the middle ear, eustachian occlusion, etc., may excite it and drugs like quinin and the salicylates may induce it.

Paralysis of the auditory nerve, resulting from a cortical lesion, may cause unilateral deafness. When the first left temporal convolution is affected by a destructive lesion *word-deafness* may ensue. The patient hears, but does not comprehend the meaning of the word. Complete deafness may result from a bilateral lesion of the temporal cortex. Involvement of the sensory part of the capsule will cause unilateral deafness with hemianesthesia on the same side. With the hemianesthesia of hysteria we may likewise detect a unilateral loss of hearing. Nuclear disease of the eighth nerve is unknown though tumors of the medulla may involve the cochlear root.

The auditory nerve may be the seat of new growths. Exposure to cold may incite a neuritis often in association with a similar condition of the facial nerve. Those who live in the midst of noise may suffer a loss of hearing in normal surroundings. Basilar fractures and extension of inflammation from the mastoid, tympanum or pharynx may be the causes of auditory disturbance. Gouty deposits and hemorrhage into the root substance have been observed. Syphilitic and purulent meningitis are also occasional causes of deafness from infectious extension into the middle ear.

When loss of hearing is due to the nerve affection it is a case of nerve deafness. When there is no involvement of other cranial nerves, when sounds of high and low pitch are not heard through the air or by bone conduction, or when there are no basilar, bulbar or cerebral indications a nerve lesion is probable. When air-conduction is better than bone-conduction we may know that the loss of hearing is a case of nerve-deafness.

The *treatment* of nerve deafness will require the services of an expert aurist. Foreign irritations and inflammatory causes must be removed if possible. Internal medicines must be used according to indications. Quinin and the salicylates must not be used. *Strychnia* is often serviceable, but should be used guardedly; *pilocarpin* has been advocated. *Electricity* may be employed with caution. The treatment usually employed for catarrhal deafness is contraindicated here.

Ménière's Disease, or aural vertigo, is an irritation of the vestibular portion of the eighth nerve. It is well known that disturbances in the middle or internal ear and many times the external meatus are liable to excite vertigo. This is most frequently observed after 30 years of age. It refers to an organic change in the labyrinth which gives rise to attacks of vertigo with nausea and perhaps syncope. This name, however, is erroneously applied to all forms of vertigo. In the true type a progressive deafness attends, while tinnitus, forced movements and even a loss of coördination may be present.

When the nerve end-organ is destroyed the deafness becomes complete and then the vertigo disappears. An acute attack of Ménière's disease may cause momentary unconsciousness, the patient often experiencing the sensation of being struck. The seizures may arise without apparent cause while again a sudden movement or a blow upon the head may induce it.

Under the subject of vertigo we may refer to other forms. *Reflex vertigo* from gastric and toxic causes is very common, inasmuch as the nucleus of the eighth nerve is connected with those of the vagi, and the sympathetic excitement may affect the circulation in the vertebral artery which supplies the labyrinth. *Lithemic* or *bilious vertigo* is created by the discharge of irritants into the blood which excite the brain. *Neurotic vertigo* may be seen in association with epilepsy, hysteria and neurasthenia. Psychical vertigo is an example of this kind. In these the patient suddenly has a sense of insecurity, an apprehension of falling, and the sense of an approaching loss of consciousness. In fact, however, there is no true vertigo, either subjective or objective. *Ocular vertigo,* caused by refractive errors and unequal action of the ocular muscles, is not common. *Mechanical vertigos* are those which are caused by seasickness, carsickness, swinging, whirling, etc. *Arteriosclerotic vertigo,* or senile vertigo, is due to perverted nutrition of the brain as the result of senile changes in the vessels.

The *diagnosis* of aural vertigo will be made only by accurate determination of the auditory symptoms; both tinnitus and defective hearing, tested by bone-conduction, will be present. If the vertigo is produced or increased by Politzerization, it is evident that the trouble is in the ear. A quick movement of the head may incite the vertigo which is usually indicative of the semicircular canal involvement. The subjective or objective gyration or fall is invariably in the same direction in aural vertigo. Epilepsy has a vertiginous aura. In true aural vertigo the sensorium is usually undisturbed except for the first momentary loss of consciousness. The sudden onset in Ménière's disease is often suggestive of cerebral disease and the vomiting which so frequently attends aural vertigo may be mistaken for a "bilious spell." When digestion is normal and there are no signs of cerebral or cardiac disturbance, and the vertigo continues either day or night, we may know that it is aural in character. The labyrinthine form of vertigo may simulate ocular vertigo, but the latter ceases when the eyes are closed, while the aural form continues.

The *treatment* of aural vertigo belongs to the specialist who is skilled in that line of work, as the Politzer inflation or even the removal of cerumen demands the experienced touch of an aurist lest damage be done. When changes exist in the labyrinth, rarely may any help be expected. Local treatments are of slight value and remedies do not generally apply in typical aural vertigo. In those due to systemic causes we may expect to give relief by our remedies. *Cocculus, lobelia, iris versicolor, ipecac, nux vomica, gelsemium, argentum nitricum, zincum picricum* and many other remedies may be used.

Disease of the Glosso-Pharyngeal Nerve.—The diseased conditions of this nerve are not fully understood. If other nerves with which it is related are affected by disease, there may be an implication of the glosso-pharyngeal. Such perversions give rise to anesthesia in the upper pharynx and in the palate. Rarely does any disease attack this nerve alone; if its nuclei in the medulla are implicated, as, for instance, in bulbar paralysis, it will account for the pharyngeal symptoms of that disease.

Diseases of the Pneumogastric and Bulbar Part of the Accessory—The *pharyngeal* branches of the vagus are paralyzed when there is nuclear disease and frequently in diphtheria. The pharynx becomes insensitive and motionless, the pharyngeal reflex being lost. A spasmodic cough or strangulation may follow from accumulation of food in the pharynx. A pharyngeal spasm is a functional feature in hysteria known as "globus," and many times simulates pharyngeal paralysis, but may be differentiated by its continuous existence. *Laryngeal paralysis* may be partial or complete and unilateral or bilateral, abductor paralysis being most frequent. In a complete bilateral paralysis the vocal cords are motionless, both recurrent nerves being affected by an injury. The voice is lost and the patient can not cough or sneeze. If a deep breath is taken stridor results. When complete paralysis is unilateral the affected cord is motionless, while its opposite is active in phonation and respiration, the voice being low-pitched and hoarse.

The *treatment* of these paralyses depends largely upon the cause. Frequently the injury of the nerve affects the pneumogastric, causing, therefore, cardiac disturbance. In case of a toxic cause internal medication according to indications, and electricity generally give relief. The purely neurotic disturbances are not long-lasting. In nuclear involvement there is no help.

Anesthesia of the larynx appears as the result of superior laryngeal nerve involvement and may be unilateral or bilateral. Hysteria and cerebral lesions may cause laryngeal hemianesthesia, while a loss of sensation at the laryngeal inlet may be found in hysterical adduction paralysis. *Laryngeal spasm* may arise reflexly from peripheral disturbance of the pneumogastric or from direct irritation of the recurrent laryngeal nerve. It may appear in connection with catarrhal laryngitis or may follow as a result of reflex disturbances from gastro-intestinal perversions. Asthma, migraine and hysteria may provoke it. When the voice has been overused a spasmodic neurosis of the larynx may result. This spasm is characterized by its sudden onset and short duration, dyspnea and stridor being temporary symptoms.

When the *pulmonary branches* are affected *spasmodic asthma* may develop. This usually arises as a result of inhaled irritants like dust or the pollen of certain plants which, by irritation of the pulmonary branches of the pneumogastric, create a spasm of the bronchioles. Intestinal and genital disturbances may also develop the spasm by the reflex disturbance of the sympathetic branches which are distributed to the bronchi. Laryngeal and bronchial irritations are also direct factors in these asthmatic attacks. The *attacks* come on suddenly and are followed by periods of complete relief from the spasm. Respiration is very difficult, expiration being prolonged. The dyspnea becomes so extreme that all the classical symptoms of asthma are experienced. These attacks and the treatment have been considered under the subject of bronchial asthma. *Hiccough,* though referred to disturbances of the phrenic nerve, is due to the laryngeal spasm and is the neurotic equivalent of asthma. Injury of the pneumogastric, or the superior laryngeal, as well as the phrenic nerve may excite it.

In the *cardiac disturbances* which arise from any involvement of the superior or inferior cardiac branches of the vagus we shall notice *tachycardia* as a most prominent symptom. This may be temporary or permanent and is "due to the increased accelerator sympathetic influence or to decreased vagus inhibitory control or to both acting together." (Church.) This usually occurs in paroxysms. It has been more fully described under cardiac diseases. *Cardiac palpitation* and *bradycardia* must also be traced to a vagus neurosis, both of which have been fully described under these special subjects.

Gastralgia or gastrodynia is a neurosis of the pneumogastric branches to the stomach, which is unattended by digestive disturbances. It may be associated with other neuralgias or migraine, and hysteria is occasionally the irritant cause. The particular features of this have been given under stomach diseases. Rumination is a symptom associated with disturbance of the gastric or esophageal branches of the pneumogastric and may be rarely noticed in hysteria, neurasthenia, epilepsy and mental diseases. *Nervous dyspepsia* must also be considered as at least a partial factor in pneumogastric disturbance. The treatment of all of these conditions has been spoken of elsewhere.

Diseases of the Spinal Portion of the Accessory Nerve.—*Accessory spasm,* better known as *torticollis,* is the result of an irritative lesion of the spinal accessory nerve. It is characterized by clonic or tonic spasm of the muscles supplied by the spinal accessory and often of other muscles of the neck. It is sometimes seen in meningitis, epilepsy and other forms of convulsions. It often exists as a *congenital wry neck* and it may appear as a symptomatic wry neck in children following a rheumatic myositis. The *spasmodic wry neck* is the typical form caused by a spasm of the muscles supplied by the spinal accessory. The *exciting cause* is not always understood. It may come from injury, undue strain of the muscles mentioned, infections, emotional excitement, etc. In my experience it more frequently arises without any known cause. The *symptoms* are characterized by the painful spasm which affects the muscle. The sternomastoid is most frequently affected, though the upper fibres of the trapezius are occasionally implicated. The head is inclined to the affected side by the action of the sternomastoid, while the chin is elevated and the face is rotated to the opposite side by the combined action of the sternomastoid and the trapezius. The head may be pulled backward when both trapezii are involved. The complexus and superior oblique may be implicated. Remote muscles may be simultaneously involved. The spasm instead of being clonic in character may be tonic. In the average case the patient suffers but little between paroxysms, though the spasm may be renewed by any nervous excitement. The disease is in reality a neurosis, the bulbar and cerebral centers being implicated. The innervation is disturbed and as a result there is an intermittent discharge of nerve force.

The *treatment* is not always satisfactory. While torticollis is not incurable it is liable to assume a chronic state which responds only to persistent treatment. By many it is regarded as an incurable disease and surgery is considered as a last resort. I am inclined to look upon the therapeutic possibility with a little more hope. I have succeeded in curing two severe cases which, fortunately, came to me in the early stages of the disease. Many *remedies* have been suggested for the treatment, among which may be mentioned ***atropin, hyoscyamus, conium, gelsemium, zinc valerianate, physostigma*** and ***agaricin.*** The last is my favorite remedy, and this I have used in conjunction with a tablet containing hydrobromate of hyoscin (gr. 1/400) cannabin (gr. 1/8) and zinc phosphide (gr. 1/10). One tablet of the latter is given four times daily. Bromids and opiates are required in the acute paroxysms, but a cure is not accomplished in this way. A remedy must be used which reduces the persistent discharge of nerve force and this will be found only after a careful study of the symptoms.

The *galvanic* and *faradic currents* may be of adjuvant service to relax the spasm, but I do not believe they are indicated in every case and their prolonged use in the interparoxysmal state may incite a spasm. Massage and vibratory stimulation are much more serviceable in the majority of cases. Nerve stretching and suspension are now properly abandoned. Resection of the posterior branches of the upper three or four cervical nerves has relieved in a few cases.

Paralysis of the Spinal Accessory Nerve.—This pertains to a palsy which affects the spinal portion of the nerve. The nuclei may be involved in a degenerative lesion which starts in the spinal gray matter. Cortical disease very rarely causes complete and permanent loss of power in the spinal accessory. The nerve-trunk may suffer compression from meningeal exudations, caries or tumors. The sternomastoid will show the paralysis while the trapezius will be slightly involved. The head is not so easily turned to the opposite side, the shoulder droops slightly and the extension of the arm is impaired. The scapula stands out from the ribs and the normal contour of the neck is lost. The lesion is usually unilateral, though a bilateral palsy may arise as the result of meningitis or vertebral caries; it may also develop in progressive muscular atrophy. If both sternomastoids are involved, the head falls backward, and if both trapezii are involved, it falls forward. The *treatment* relates to the underlying cause; in nuclear lesions no hope may be expected.

Diseases of the Hypoglossal Nerve.—The hypoglossal nerve is the motor nerve for the muscles of the tongue and those attached to the hyoid bone. *Spasm* of the hypoglossal nerve takes place in hysteria, epilepsy and sometimes in chorea. Irritation of the fifth nerve is also responsible for the spasm. A paroxysmal clonic spasm is a form characterized by the rapid protrusion and withdrawal of the tongue. The prognosis in these cases is good and the treatment rests on the use of the indicated remedy. *Paralysis of the hypoglossal nerve* is the result of supra-nuclear, infra-nuclear and nuclear lesions. In the *supra-nuclear paralysis* the lesion is between the cortex and medulla, the paralysis appearing on the opposite side. Atrophy and electric changes are not noticed. In *infra-nuclear paralysis* the lesion may be due to the pressure of neoplasms or meningeal or syphilitic exudates. *Nuclear lesions* are usually degenerative.

The *symptoms* are characterized by paralysis and atrophy of the tongue on one or both sides, fibrillary twitchings and, in unilateral paralysis, the tongue deviates to the paralyzed side. Articulation, mastication and deglutition are slightly interfered with. In the bilateral form these perversions are marked and the tongue can not be protruded. When the lesion is supranuclear there is hemiplegia on the same side as the lingual paralysis. When the lesion is nuclear a bulbar palsy may exist. When the medulla fibres are involved paralysis of the tongue will take place on one side, while the limbs will be paralyzed on the opposite side. Outside of the medulla the spinal accessory nerve is generally involved and the paralysis is unilateral.

DISEASES OF THE SPINAL NERVES.

Diseases of the Cervical Plexus.—The *phrenic* nerve may be affected when there is a lesion of the ganglion cells in the anterior horns near the level of the third and fourth cervical nerve. The trunk may be affected by neuritis, traumatism or by the pressure of an aneurysm or neoplasms. The diaphragm is always involved when this nerve is implicated and this affection will vary between a complete paralysis and immobility of varying degrees. In such instances diaphragmatic action shows reversed movement. Exertion causes dyspnea and pulmonary diseases are inclined to be exaggerated when there is diaphragmatic immobility. Other muscles are liable to be affected when the diaphragm is implicated.

Diseases of the Brachial Plexus.—The plexus as a whole may be involved or single branches may be affected separately. A disturbance may likewise be confined to the nerve-roots which unite to form the brachial plexus. Meningeal disease, traumatism and caries of the vertebrae may constitute the causes. The symptoms are usually unilateral and purely sensory and motor in character unless the lesion is extensive. The *posterior thoracic,* if affected, will show its symptoms in the serratus magnus muscle. The posterior edge of the scapula will stand out prominently and this will be exaggerated when the arm is moved forward. Neuralgic pains are generally pronounced and the neuritis pursues a slow course.

Combined paralysis may be experienced when the brachial plexus is involved and this may result from the involvement of two or more nerves or the plexus as a whole. New growths in the cervical region, wounds, fractures and dislocations are the usual causes. A brachial neuritis may be associated with an injury to some nerve communicating with it, or it may be a primary affection. A gouty diathesis predisposes. The paroxysmal or continuous pain is increased by any sudden movement or local pressure. Sleep-palsy, or crutch-palsy, are samples of the pressure-palsies which frequently occur.

Suprascapular palsy is observed when, by the involvement of the supra and infraspinatus muscles, the outward rotation of the humerus and the rotation of the scapula is interfered with. The deltoid naturally hypertrophies on account of the extra work demanded of it. When the *circumflex* is involved the arm can not be raised at right angles to the body and the involved muscles, the deltoid and teres minor suffer atrophy.

Musculo-spiral paralysis is very common, as it is particularly exposed to pressure. The characteristic symptoms are wrist-drop, finger-drop and inability to extend the hand, the phalanges of the fingers or the thumb. Supination is also frequently lost in pressure-palsies. There are only slight sensory symptoms. Lead-palsy is differentiated by its slower onset, absence of supinator involvement and its bilateral character. Besides there is usually a history of lead-poisoning. Bilateral wrist-drop in toxic neuritis is generally differentiated by the manner of the attack and the history of the case.

The *median nerve* may suffer from neuritis following an injury, but is generally affected in association with other nerves. Wasting of the thenar muscles, inability to oppose the thumb to the tips of the fingers, imperfect flexion of the phalanges and the radial side of the wrist are the characteristic symptoms. *Ulnar paralysis* causes radial deviation of the hand on flexion of the wrist, loss of abduction of the thumb and inability to move the little finger. The hypothenar prominence suffers from wasting. The first phalanges can not be flexed and the second and third can not be extended. The "claw hand" is usually detected, but it is not so pronounced as in progressive muscular atrophy, as the median supply overcomes this extreme change.

DISEASES OF THE LUMBAR AND SACRAL PLEXUSES.

These plexuses are involved occasionally by abdominal growths, enlarged glands, psoas abscess, neuritis, vertebral diseases and occasionally by wounds and dislocation of the hip. The *obturator nerve* is affected when adduction of the thigh is interfered with, outward rotation is somewhat impaired and the affected leg can not be crossed over the other. *Anterior crural nerve* paralysis is recognized by the loss of power and wasting of the leg extensors. Lost knee-jerk and anesthesia of much of the surface of the thigh and the inner side of the leg and foot may also be noticed. The *superior gluteal nerve,* if paralyzed, shows a loss of adduction and circumduction of the thigh.

In *sacral plexus* paralysis the *small sciatic,* when involved, causes difficulty in rising from the sitting position, and anesthesia of the back of the thigh and of the upper part of the leg posteriorly. When the *great sciatic* is involved, the act of walking will be interfered with and anesthesia and wasting of the flexor muscles of the legs and the muscles below the knee will be observed.

When the *external popliteal* or *peroneal nerves* are involved there will be foot-drop and toe-drop and the region supplied by these nerves will be anesthetic. Involvement of the *internal popliteal nerve* causes inability to flex the foot and toes, and anesthesia over the area of distribution.

DISEASES OF THE SPINAL CORD.

SPINAL HEMORRHAGE.

Hemorrhages affecting the meninges of the cord and the cord proper are generally classed together so far as treatment is concerned, therefore we consider them under the term of spinal hemorrhage.

Spinal Meningeal Hemorrhage.—This is the common form of spinal hemorrhage. It is either extradural or subdural. It may be associated with intracranial hemorrhage or with hemorrhage into the cord substance. It also occurs as a primary condition.

Etiology.—Injuries, particularly spinal fracture, are the most common exciting causes. Diseases of the meningeal vessels may produce it; syphilis, arteriosclerosis, purpura and all infections and dyscrasiae naturally predispose to it. Severe convulsions, epilepsy, eclampsia, tetanus and chorea may favor it. The rupture of the thoracic or vertebral aneurysm or a prolonged meningitis may also be exciting factors. It may be due to severe muscular exertion or it may rarely occur spontaneously.

Pathology.—In *extradural hemorrhage* the clot is generally located in the cervical region; it may be small or large and may extend through the intervertebral foramina. The dura may be infiltrated for a considerable area and cord compression in varying degree may occur. The hemorrhage comes from the vertebral plexus of veins. Hemorrhage *within the dura* may be extensive or slight, the central arteries being its source. Cord compression and meningeal inflammation generally attend.

Symptoms.—The signs of meningeal hemorrhage usually appear suddenly. In small hemorrhages there may be no confirming evidence. The symptoms of extra- and subdural hemorrhage are virtually the same in the beginning. *Severe pains* in the back, in the neighborhood of the hemorrhage, are first experienced, with backache and numbness for several hours. These pains extend along the nerves the roots of which are involved. *Rigidity* of the spinal muscles is characteristic and may reach the stage of opisthotonos and convulsions. *Numbness,* tingling, formication, hyperesthesia and paralytic symptoms affecting the muscles below may follow with loss of control of the bowels and bladder. *Anesthesia,* together with other visceral disorders, may now attend the paralysis. A chronic meningitis or myelitis may ensue and the recovery will be slow.

Diagnosis.—A *well defined onset* and the gradually developing secondary symptoms, with the history of traumatism, ought to confirm the diagnosis; insidious symptoms may confuse. When the *cord substance* is affected (as in hematomyelia) there is instant loss of function and subsequent anesthesia; the pain is less but the paralysis is worse. In *meningitis* the symptoms do not develop so rapidly and the cause is not so apparent. From *tetanus* we differentiate by the trismus in the latter disease.

Prognosis.—An unfavorable outcome is probable in most cases; immediate death is possible. If the patient survives a few days, a partial recovery may be looked for. Subsequent inflammatory invasion and respiratory paralysis may cause a fatal termination. Hemorrhage in the cervical area is most dangerous.

Treatment.—*Absolute rest* in bed and perfect relaxation are mandatory. Local application of the *icebag* to the spine will often give relief; should it increase the irritation it must be discontinued. The patient should be given only a liquid diet and complete catharsis should be effected. The enema and the catheter should be resorted to to avoid any possible exertion. Cupping, leeches and venesection have been suggested by older authors.

The remedies which aid the absorption of the blood clot should be given internally. *Secale* is of service in the early stages to check the hemorrhage; *hamamelis, ipecac, hydrangea, erigeron* and similar remedies may be indicated. *Aconite, arnica, belladonna, gelsemium* and *hypericum* generally suffice for the acute febrile stage.

Later, to encourage absorption, the iodids, mercurius, sulphur, nitric acid and similar remedies may be indicated. The more complete treatment will be considered under hematomyelia.

HEMORRHAGE INTO THE CORD.

Etiology.—Hemorrhage into the cord substance, hematomyelia, is comparatively frequent, occurring as the result of severe spinal concussion, violent muscular wrenching, injuries, exposures, and sometimes from excessive coitus. Again it may be secondary to diseases of the blood vessels, purpura, syphilis, myelitis, tumors or convulsions. The hemorrhage is frequently located in an area of myelitic softening, though the latter may also follow the hemorrhage. In old people degeneration of the arteries is more frequently the cause of this disease.

Pathology.—The hemorrhage is punctate and multiple, or it may be single and extensive; it occurs in the central arteries and, hence, is found mostly in the gray matter of the cord; it may penetrate the white columns or, less commonly, the central canal. In the punctate variety small pinhead clots are found in the slightly reddened cord; they are mostly in the perivascular spaces. The single or larger hemorrhage invades even more of the gray matter, causing a decided swelling of the cord. A zone of softening is observed about the clot a few days after the hemorrhage, which may develop into a more extensive myelitis.

Symptoms.—*Numbness* and *weakness* in the limbs are the first premonitions, then *paraplegia* appears suddenly, which is the first sign of a primary focal hemorrhage. The extent of the paralysis depends upon the size of the clot. *Anesthesia* is a characteristic symptom, the tactile sense is preserved, but the pain and thermic sense are lost. The *sphincters* are paralyzed, the reflexes are at first lost, but soon become exaggerated and ataxia may supervene. If the lesion is in the upper part of the cord, the arms may be affected also. In about ten days the acute symptoms subside and the disease then simulates chronic myelitis.

Diagnosis.—We may differentiate between this and *meningeal hemorrhage* by the fact that the latter shows more pain and spasm, less paralysis and has a greater tendency to recovery. Hematomyelia has a more sudden onset and less fever; often the symptoms are so similar that it is difficult to make a perfect diagnosis.

Prognosis.—This depends upon the extent of the hemorrhage, the subsequent softening and the location of the lesion. In the cervical region, it is almost invariably fatal; in the lumbar region it is unfavorable, while in the dorsal region the prognosis is more hopeful. If the secondary myelitis extends upward, it causes certain death, if it extends downward, it enhances the paraplegia. Permanent paralysis, pronounced trophic disturbances, spastic ataxia or an incurable chronic myelitis are possible results of the hemorrhage.

Treatment.—At first it is necessary to insure absolute rest and quiet. The patient must be kept in bed for several weeks in a position that will cause the least pressure upon the spine. The ice bag applied to the involved area gives much relief. The diet should be limited and the bowels should be relieved by means of an enema.

Remedies which reduce arterial tension and control the hemorrhage should be employed. *Aconite* and *veratrum viride* are the most serviceable in the first stages; *belladonna* answers well in plethoric patients; *secale,* in potency, may be used for a time, but heroic doses of ergot should not be employed. *Hamamelis* and arnica should not be overlooked when traumatism is a causative factor. *Ipecac* will be of service when gastric disturbances attend. *Trillium, sabina, geranium, erigeron* and *ustilago* are useful, if indicated, the same as in any other hemorrhage. *Arsenicum* may be indicated when the hemorrhage is attended by asthenic conditions. *Cinchona and ferrum phos.* are called for when anemia is present. *Oxalic acid* is useful when exhaustion is attended by severe pains extending up and down the back. *Nitric acid* and *mercurius* may be demanded as the condition approaches the chronic stage. *Opium, codein* and *adrenalin chlorid* should not be overlooked.

When the danger from hemorrhage and its attending symptoms disappears, the treatment and the remedies which apply in chronic myelitis will be demanded, therefore, further discussion of the treatment will be found under that subject.

CAISSON DISEASE.

This is a disease which occurs with those who are subjected to high atmospheric pressure. It is observed most frequently with men who work in caissons, hence, its title. The symptoms appear when there is a sudden return from a condensed air to the normal atmosphere. Aeronauts, mountain climbers and workers in high altitudes may suffer with a mild form of the disease.

Etiology.—Those whose vocations accustom them to unusual atmospheric pressure do not ordinarily experience any discomfort; the novice is more liable to this disease. The trouble comes mostly from the sudden reduction of the pressure. Age, alcoholism, obesity, heart and kidney diseases predispose to its occurrence.

Symptoms.—These usually appear very soon after the workman reaches the surface. Sometimes they do not appear until a few hours later. *Headache,* dizziness, faintness, nausea, vomiting, delirium and often coma are early symptoms. The spinal symptoms are most severe. *Paroxysmal pains* in the lower extremities are most intense, and while the legs are generally affected more frequently, the pain may involve the arms as well. Anesthesia attends the weakness and soon *paraplegia* develops. Hemiplegia is an infrequent symptom and the sphincters are generally paralyzed. The *duration* of the disease is not long. With favorable physical conditions a speedy cure is possible. Amelioration of pain comes slowly, while the paraplegia may remain for some time or may become permanent.

Pathology.—When the atmospheric pressure is increased, the capillary system becomes paretic, the internal viscera are congested and thus a brain and cord hyperemia exist. When the workman returns to the normal atmosphere, blood rapidly rushes to the surface, causing a peripheral congestion. The brain and cord, being in bony cavities, can not rapidly eliminate the excessive amount of blood. As a result hemorrhage or a permanent congestion is found in these parts. Following this, there is a liability to persistent inflammation or necrosis of nerve tissue, particularly in the cord.

Treatment.—This is mostly expectant and symptomatic. Only a perfectly sound man should be permitted to assume this kind of work. More time should be taken to return to the surface. When the disease exists we should put the patient into the most perfect state of rest and relaxation. Morphin and chloral may be necessary to relieve the pain and nervous excitement. The treatment of the paraplegia will be the same as given under myelitis.

SPINAL HYPEREMIA.

This is a disease which is really a symptom present in the first stages of acute inflammations. It is incident to violent physical exertion, sexual excess, menstrual disturbances, and toxemia, and may be acute or chronic. The latter form, though not well defined, may follow meningitis or injury.

Pathology.—This is not marked, as the disease is simply a congestion of the spinal vessels and may depend upon exaggerated or diminished cardiac action.

Symptoms.—Neuralgic pains, numbness and other paresthesiae appear early; a feeling of heaviness and of weight in the limbs and back are then experienced: Twitching of the muscles and slight sphincter weakness may be complaints in the early stages. The weakness of the limbs may disappear in a short time or it may persist. Generally speaking, the symptoms are mild in character and short in duration.

Treatment.—This condition is generally cared for successfully by the remedies indicated in acute febrile conditions. *Aconite, belladonna, cimicifuga, gelsemium, arnica, hamamelis* and *millefolium* are usually sufficient. *Arnica, nitric acid* and *china* are good secondary remedies.

The patient should be kept very quiet and in a horizontal position for some time. The ice bag, applied to the spine, will invariably give relief in the early stages. Excessive pain will demand an opiate, while the chronic stage may call for the galvanic current. Perfect rest and the internal remedy are about the only therapeutic measures necessary.

SPINAL ANEMIA.

Whenever the circulation is interfered with, the spinal cord is liable to suffer from anemia. Hemorrhages, exhausting diseases and diseases affecting the aortic valves of the heart are sources of this trouble. When spinal anemia exists the cord functions are greatly diminished, with a resultant impairment of motor function; weakness of the legs amounting to apparent paraplegia is experienced, particularly after an undue amount of exercise. There may be periodic pains in the back, especially when the patient suffers from fatigue. Spinal irritation often is a residuum and requires careful treatment. In other respects the symptoms resemble neurasthenia. The treatment is purely symptomatic and the prognosis is generally favorable. Freedom from nervous excitement and the worry and demands of business is mandatory. These patients should forego all social indulgences and a treatment of systematic rest must be instituted.

SPINAL MENINGITIS.

Pachymeningitis Externa.—**Etiology.**—This is always secondary to some other disease. Tuberculosis and spinal caries are the most frequent causes, while purulent pleurisy, peritonitis, mediastinal collections of pus and even pelvic abscesses may be more rarely the primary factors.

Pathology.—The dura is thickened and covered by a caseous or purulent deposit, the posterior aspect of the cord being most involved. The inflammatory exudate, when acute, is fibro-purulent in character when chronic, connective tissue predominates, and this may affect the cord and all the membranes. In such cases a compression myelitis is liable to follow.

Symptoms.—*Radiating pains* indicative of an irritation of the sensory and motor nerve roots of the part affected are early symptoms. Hyperesthesia, twitchings, exaggeration of reflexes, paraplegia and sphincter perversions are subsequent symptoms. Anesthesia may occur later, or it may be an early sign in severe cases. When cord compression follows from unusual thickening, an extreme spastic paraplegia is observed.

Diagnosis.—This is aided decidedly by determining the predisposing disease. The pronounced radiating pain, the local tenderness and irritability, the chronic course and the paralysis from compression late in the disease, should settle the diagnosis. *Myelitis* should be differentiated by the rapid onset, the slight amount of pain, the girdle sensation, the anesthesia below this point, the sudden paralysis and the pronounced involvement of the sphincters.

Prognosis.—This is almost always unfavorable. If compression does not exist and the case is energetically treated, we may avoid the protracted chronic condition.

Treatment.—The treatment is most decidedly surgical, inasmuch as the inflammation is usually due to mixed infection. Mechanical, hygienic and constitutional treatment will do much for these cases.

Pachymeningitis Interna.—This includes two forms, the hemorrhagic and hypertrophic. The hypertrophic form represents simply a thickening of the exudate due to hemorrhage. The two forms are, therefore, included in this discussion.

Etiology.—It occurs most frequently in adults. Exposure and traumatism, alcoholism, syphilis and other infections are the most common causes.

Pathology.—The primary pathologic condition is a hemorrhage upon the surface of the dura, followed by chronic inflammation and recurrent hemorrhages, resulting in an exudate of fibrous tissues which soon encircles and compresses the pia mater and cord. This exudate may be extensive, but usually it is confined to the cervical area. When syphilis is the cause, the exudate is gummatous in character.

Symptoms.—The disease is a chronic one, and therefore the symptoms are slow in their development. Irritation of the nerve roots gives rise to hyperesthesia and pain radiating along the spinal nerves, which have their origin in the spinal segment involved; these pains exacerbate at night and considerable stiffness in the neck is observed in the beginning. Numbness, prickling and pain are felt in one or both arms. Gastric crises are sometimes observed.

The *paralytic symptoms* are apparent a few months or a year later. There is a gradually developing weakness in the parts involved, while anesthesia and trophic changes slowly make their appearance. As the cord compression ensues, *spastic symptoms* are observed in the parts supplied by the affected nerves. *Rigidity,* exaggerated reflexes and spinal trepidation are followed by paraplegia; this condition may remain stationary for some time and then recovery may take place. Frequently one or both arms are affected, which depends upon a meningeal invasion of a small vertical area in the cervical region and is termed, *pachymeningitis cervicalis hypertrophica.* This form gives a peculiar deformity; the flexor muscles are most affected, particularly the small muscles of the hand and arm. The fingers are forcibly flexed and the hand is often extremely extended. In some cases the ulnar nerve is affected most and a peculiar wrist drop exists. The disease is liable to affect the general health and strength, and the patient may die from exhaustion or some other intercurrent trouble.

Prognosis.—Little hope of a cure should be given. The syphilitic cases are most unfavorable, but those caused by a slight trauma are more hopeful. The disease may assume a stationary condition for a long time.

Diagnosis.—The diagnosis is most difficult when the inflammatory exudate is extensive. The chronic course, the characteristic pain and the localized symptoms should lead to the diagnosis. *Tumor of the cord* is most simulating, though the symptoms are more localized and it does not run so chronic a course. *Myelitis* more frequently affects the lower cord, and the paralysis is acute. The anesthesia, the sphincter involvement and other characteristic symptoms should also aid in the differentiation. In *progressive muscular atrophy* the muscular degeneration is the cause of the paralysis and other symptoms.

Treatment.—The remedies suggested previously for hemorrhage and febrile conditions should be used in accordance with indications. Hydrotherapy, and the applications of dry heat may palliate in the beginning. Massage and electricity may relieve the rigid muscles, but the paralysis will not be cured unless the exudate is absorbed and the function of the compressed nerves is restored. The syphilitic cases must be treated with consideration for the cause. In general, *potassium iodid, chlorid of gold, iodid of arsenic, nitric acid* and *mercurius* will be standard remedies. For the muscular pains and weakness of the limbs *gelsemium, cimicifuga, conium, cedron, china* and *cicuta virosa* should be considered. *Plumbum iodid* is of great service when the pathologic condition tends to sclerosis and when there is glandular induration. *Iodid of* *antimony* and the *iodid of tin* have been suggested in the chronic stages; *zinc picrate* and the *phosphid of zinc* will always be worthy of consideration.

ACUTE SPINAL LEPTOMENINGITIS.

Etiology.—This is generally observed in connection with inflammation of the cerebral pia mater. It is due to infection and it may involve the inner surface of the dura and the cord periphery as well. Typhoid, tuberculosis, syphilitic and other infections may be the cause. The pneumococcus and the meningococcus have been found as etiologic factors. Traumatism, exposure and rheumatism are predisposing causes. Surgical operations and penetrating wounds may furnish an infection if asepsis is not perfectly observed.

Pathology.—The pathology depends much upon the character of the exudate; the suppurative exudate is the most characteristic. The infection is easily propagated and transmitted through the spinal fluid of the arachnoid space. The pia, the inner surfaces of the dura and the cord periphery are hyperemic and the spinal fluid is increased in quantity. If the inflammation is protracted, the connective tissue increase causes dural, arachnoid and pial adhesions; the arachnoid spaces are thus obliterated and compression of the nerve roots and the cord creates structural changes. We may find degeneration of the fiber tracts whenever the softening process occurs. In tubercular infection the exudate is not so excessive and resembles fibrous tissue.

Symptoms.—The disease is ushered in by *chills* and *fever,* often preceded by malaise and gastric disturbance. *Tenderness* and pain in the back now appear and muscular rigidity soon is noticed; this may give rise to convulsions and particularly opisthotonos. *Dyspnea,* Cheyne-Stokes' respiration and other serious symptoms may develop rapidly. Hyperesthesia gives place to anesthesia and eventually paralysis with sphincter involvement takes place.

Prognosis.—The outcome of this disease is rarely favorable, while the tuberculous form suggests a still less favorable prognosis. A chronic form of meningitis may be a bothersome sequela.

Diagnosis.—There is not a great amount of confusion in making the diagnosis when we consider the essential signs of infection. *Gonorrheal rheumatism* gives the most complete symptomatic resemblance, but this should be easily differentiated.

Treatment.—The general and medicinal treatment does not differ materially from that of pachymeningitis. The importance of absolute rest and quiet must not be overlooked. Hot applications or cold applications should be employed; the ice bag will, on the average, give better relief than anything else. Counter-irritation, cupping and leeches are still advocated in accordance with the old line of practice, but the necessity of these measures is not established. In the plethoric patient, bleeding may be indicated, and in certain cases we may find counter-irritation of some service. If, however, we assist nature by prescribing for actual symptoms and keep the patient perfectly quiet, we shall get better results.

As the disease progresses we may sometimes find efficacy in the use of the hot spinal spray, but the old theory of spinal cauterization is obsolete. When cerebral symptoms appear we shall find use for such remedies as *aconite, belladonna, hyoscyamus, stramonium and anacardium.* For the paralytic conditions which result from acute leptomeningitis we should resort to the adjuvant treatment of electricity, massage, etc., the same as for other forms of paralysis.

CHRONIC SPINAL LEPTOMENINGITIS.

We do not often observe this disease except as it occurs as a sequel of the acute form. Alcoholism and syphilis may incite it, but even these causes are more liable to be factors in developing acute symptoms. Traumatism and spinal concussion may create a slowly developing inflammation which partakes of a chronic character.

Pathology.—Extensive fibrous thickening of the pia mater and the arachnoid, may extend to the cord and cause a ringlike sclerosis; the degeneration may then involve isolated parts of the cord until certain portions of the fiber tracts are affected. This is better defined as meningo-myelitis. The nerve roots found in this sclerotic mass may suffer a like degeneration and as a result of this we are liable to find herpetic and other cutaneous symptoms.

Symptoms.—These resemble those of the acute form, but they are not so pronounced in intensity. Tenderness along the spine, and radiating pains are present, which develop slowly and are not constant. There is some spasm and twitching of the limbs and a slight palsy may develop after a time. Anesthesia occurs later and trophic changes may be marked. There are periods of improvement, while all symptoms are relieved by absolute rest.

Diagnosis.—A careful study of the symptoms and their cause should settle the diagnosis. Spinal irritation may have some similar symptoms, but they are not so extreme, as the pain, rigidity, atrophy and paralysis are not so pronounced.

Treatment.—When the disease is a residuum of the acute form, the treatment will demand the management described under that disease. For special remedies we should be guided by the indicated symptoms.

MYELITIS.

By the term myelitis we refer to an inflammation of the spinal cord. The pathologic condition resulting from this inflammation may assume various forms. For instance, we speak of *diffuse myelitis* when it involves, to any extent, both the gray and white substance of the cord. *Transverse myelitis* describes a sectional inflammation of the cord and is the usual form which we meet. *Anterior poliomyelitis* refers to the involvement of the anterior horns. We find many other terms used to express the character of inflammation and subsequent softening. For instance, *hemorrhagic myelitis* refers to the hemorrhage which creates it; *compressive, septic, purulent, tuberculous and syphilitic myelitis* are all named from the specific cause.

Acute Transverse Myelitis. Etiology.—This form defines the ordinary case of myelitis; it may have a variety of causes and attending conditions, and the pathologic change may involve only a small portion of the cord, or may be quite diffuse. Ordinarily we speak of it as an inflammatory invasion, followed by acute softening, which affects a vertical section of an inch or more and extends completely across the cord. The character of the infection and the course and the extent of the inflammation will define the morbid anatomy.

A *predisposing cause* may be found in the neuropathic tendency; any previous disease of the nervous system may encourage it, but hemorrhage is the most frequent and direct cause. Injury, violent muscular effort, spinal concussion, hemophilia, syphilitic meningitis and possibly cold and exposure are conducive to its onset. Extension of inflammation from the bony structure and surrounding tissues, the meningeal inflammations and all the acute infectious diseases are factors.

Pathology.—The primary process is inflammation, but whether this precedes or follows the hemorrhage, it is difficult to state. Hemorrhagic extravasation, with subsequent softening, is the next pathologic stage. The section affected is swollen, soft and hyperemic, the cord structure being considerably disintegrated with granular, amyloid, fatty and blood cells in evidence. Spider cells (Deiter's) are usually increased in number. The axis cylinders and their sheaths are swollen, while the perivascular spaces are filled with hemorrhagic exudate and leukocytes.

The granular matter and the nerve detritus increase as the disease progresses. Connective tissue changes soon affect the white matter until the cord becomes vacuolated. The degeneration may extend up and down, involving different fiber tracts or, in the typical transverse form, it extends directly across the cord, causing a complete paralysis of parts below. The foci of softening may appear in isolated parts of the cord structure, creating a disseminated myelitis; cellular infiltration may also dilate or obliterate the central canal. It is possible in extremely acute cases that the suppurative process may be sufficient to create abscesses and in such cases death rapidly supervenes.

Symptoms.—While we find certain *cardinal symptoms* in myelitis, their characteristic totality is dependent upon the location and extent of the lesion. The onset varies and in some cases, particularly in hemorrhage, the symptoms suddenly appear. In other cases there are prodromal signs of paresthesia or pain in the back. The infectious causes develop primary symptoms in accordance with the causative disease. Convulsions, high fever and rigors usually indicate a grave condition.

The *initial symptoms* start with a feeling of numbness and weakness in the legs. Pain, which is sometimes sharp and lancinating, is felt in the back and may girdle the trunk. Following this, suddenly in hemorrhagic cases and slowly in infectious cases, *paraplegia* is observed; if the lesion is in the cervical cord, the arms will be affected. *Anesthesia* and *sphincter paralysis* usually soon follow. The febrile conditions depend upon the infection.

The symptoms of *an ordinary attack* are about as follows: The patient notices a pain in the back, numbness and a sense of weight in the limbs; he is not able to walk or use the limbs perfectly. A girdle sensation is felt at the level of the spinal lesion. Anesthesia to touch, pain and temperature exists in parts below the lesion; this is greatest so far as touch and temperature are concerned. There is vesical anesthesia and the rectal and bladder paralysis ensue. A catheter is necessary to draw the urine and the fecal matter must be removed by enema or mechanical means. When hemorrhage is the direct cause, these symptoms appear suddenly. The flexors are involved more than the extensors. The order of the symptoms depends upon the portion of the cord involved and the destructive nature of the infection and inflammation. When the posterior nerve roots and the meninges are involved, the local pain and tenderness will be excessive. In general the pain is less than in meningitis.

The *reflexes* below the part affected are generally diminished at first; in a few days or weeks they gradually become exaggerated. In lumbar lesions the skin and tendon reflexes are weakened for some time and the paralysis is flaccid. When the lesion is transverse and complete the flaccid palsy remains for some time, but there generally are sufficient motor fibres preserved to allow the development of rigidity later. In *the typical case,* after the early symptoms have disappeared, the muscles affected are rigid and considerable spasticity is present. The limbs are rigidly extended and adducted. Contractures may develop later. The sexual power is lost when there is lumbar involvement, while priapism may result from cervical lesions.

Trophic changes appear as the disease progresses. The muscles supplied by the nerves affected begin to atrophy and show the reaction of degeneration. The limbs waste from inaction and the general trophic functions are depleted. The skin is dry and unhealthy and bed sores are common. After a few weeks, if the patient lives, the disease becomes stationary. When there are signs of improvement, the sensations gradually return. A little later slight motor power is observed in the affected muscles, but spasms and contractions, however, are liable to be greater at this period. As the patient begins to use the limbs, the gait assumes that of "ataxic paraplegia."

Diagnosis.—The diagnosis is made only after a careful consideration of the foregoing symptoms. The sudden onset, particularly if hemorrhage is a cause, the early radiating pain, the distribution, extent and character of the paralysis, the sphincter involvement, the girdle sensation, the anesthesia below the point of the lesion and the hyperesthetic zone just above this, are symptoms which will rarely fail as guides to a correct diagnosis. In *poliomyelitis* sensory disturbances are not pronounced and the paralysis is liable to be monoplegic. In *meningitis* pain is more decided. *Spinal hemorrhages* are more sudden and have less fever. *Multiple neuritis* has a slow onset, affects the extensor muscles of the four extremities and paresthesia is pronounced. *Hysteria* should be differentiated by the stigmata, the characteristic hemianesthesia and hyperesthetic *zones,* and the absence of trophic changes. *Landry's palsy* (an acute ascending myelitis) shows a steady paralytic advance from the legs upward.

Prognosis.—The outlook for a cure in this disease is decidedly unfavorable. If the patient does not die during the acute attack, the paralytic sequences are liable to take him off later. Of course, everything depends upon the extent of the paralysis. The prognosis is generally worse in cervical myelitis, less dangerous in lumbar and least dangerous in dorsal myelitis. At best the case may reach a chronic stage in which the patient may be fairly comfortable if he is not absolutely cured.

Treatment.—This must consider the cause, the symptoms of the attack and the subsequent paraplegia. The *cause* must be constantly kept in mind, though this may not be the guiding requirement in the beginning. Our first attention is called to the features of an attack; the patient generally is suddenly paralyzed and we learn only of the predisposing symptoms later. The inflamed cord should be quieted and soothed by perfect rest and any external application which will relieve the congestion. *Hot fomentations* or the *ice bag* will answer this purpose. The *horizontal position* of the patient must be maintained and the back should be elevated so that no pressure occurs at the point of the lesion. The theory of systematic cartharsis is right, as it corrects the blood pressure and aids the bowel movement; generally, however, the peristaltic power is so perverted that mechanical means are necessary for evacuation of the bowels.

The *temperature* is to be corrected by remedies which are indicated by the symptoms and the character of the infection. When first called to the case *secale, arnica, ipecac, convallaria, hamamelis, phosphorus, hydrangea, opium* and *digitalis* should be considered with reference to the hemorrhage as a cause. Any other remedy or expedient indicated for hemorrhage or the prevailing symptoms should be resorted to. The fever should be treated with reference to the infection which creates it. *Baptisia, china, arsenicum, rhus tox* and *echinacea* will be found most serviceable. For the pain *bryonia, gelsemium, cimicifuga, mezereum, cedron, cicuta virosa* and *caulophyllum* will be frequently indicated. *Aconite, belladonna* and *veratrum viride* should not be overlooked in the early febrile stage. *Hypericum, dulcamara* and *ruta graveolens* should be studied in the early stages. The bladder symptoms will often suggest *hyoscyamus, cannabis sat.* or *benzoic acid.* The systemic infection may demand a limited diet, the tepid sponge and the use of an internal antiseptic. Traumatism, the meningeal hemorrhages or the presence of any new growth may necessitate surgical procedure.

The *later stages of myelitis* are most discouraging, for the improvement, if there is any, is very slow. Careful massage, faradism and possibly galvanism may now be employed. Some times the spasticity becomes so severe that splints are necessary to prevent contractures. The fulgurating pains may be relieved by the use of morphin. Bed sores demand the most careful attention on account of the necrotic tendency; a protected air or water cushion must be continuously used to protect these parts; a gauze pad and dry dressings will aid the local recovery; often it is necessary to use iodin, balsam of peru, carbolic acid or the actual cautery to obtain healthy granulation; the tar plaster may also be used as a constant dressing in these suppurative sores.

Cystitis is such a serious complication that alkaline drinks, bladder irrigation and the use of urinary antiseptics may be necessary to supplement the internal remedy, as catheterization is required for a long time, and the most strict asepsis is essential. When the urine begins to come involuntarily, a urinal must be attached constantly.

There are several remedies to be considered in the secondary stage. *Mercurius, lachesis* and *sulphur* always refer to constitutional disorders. *Oxalic acid* and *nitric acid* relate to the secondary conditions arising from hemorrhage. *Kali iodid, mercurius iodid* and the *chlorid of gold* have reference to specific causes. The degeneration of nerve tissue will call for *phosphorus, phosphoric acid, natrum phos., argentum nitricum, plumbum,* the *zincs* and many other similar acting remedies. My preference is for the continued use of the zincs, and of these I obtain best results from the *picrate of zinc.* Should the patient recover sufficiently to use his limbs in the least he should be taught how to use them so as to encourage a return to normal motor power. During all this time the aim should be to nourish and build up the depleted system.

CHRONIC MYELITIS.

Etiology.—The term chronic myelitis serves to define the conditions which follow the acute attack. It pertains to the paraplegia and the other symptoms which naturally appear when the inflammation is subacute, and when the signs of both degeneration and repair are present. It includes several forms which represent the character of the softening. For instance, we speak of a diffuse or disseminated, central or marginal form, in accordance with the location of the disease. *Compression myelitis* has already been referred to.

When myelitis relates to the chronic stage following the acute form, it is considered as a secondary disease. When it develops independently, and slowly, it is termed *primary myelitis.* This form may occasionally come from exposure, injury, infections, lead poisoning and particularly from syphilis. Rarely we find meningitis and neuritis as causes.

Pathology.—The cord and meninges are indurated in the affected part. The nerve structure, at this point, becomes atrophied, connective tissue is abundant and there is an increase of blood vessels. The morbid condition shows various stages of inflammatory degeneration.

Symptoms.—The signs of chronic myelitis are slow in development as contrasted with the acute form. The patient experiences numbness in the limbs and feet, while weakness and heaviness of the limbs are noticed after slight exertion. *Slight pain* may be felt in the back and a sense of constriction is complained of. Later the *limbs become rigid* and locomotion is imperfect; the knee reflex is exaggerated and ankle clonus may be found. The sexual and the sphincteric functions are naturally weakened. The anesthesia is not pronounced, but a slowly developing *paraplegia* appears a month or two later.

The above symptoms *progressively increase in severity,* the involved muscles begin to atrophy, the sphincters are less active and the paraplegia reaches a state of more complete paralysis. If the patient is able to get about, he shows a rigid, imperfect and shuffling gait. When the paraplegia is complete, anesthesia, contractions and pronounced atrophy make him a hopeless paralytic. Then the vasomotor perversions are more rapid in development and bed sores and other signs of trophic depletion are observed.

The *character of the lesion* varies the symptoms, the paraplegia being worse when the lumber segment is involved. When the lesion is in the cervical area, the arms are affected and the respiratory muscles may be paralyzed. *Compression myelitis* gives radiating and more pronounced pain, dependent, of course upon the amount of pressure on the spinal roots. When the dorsal region is affected the sphincter involvement is not so extreme. *Central myelitis* is difficult to determine inasmuch as none of the cardinal symptoms of myelitis are pronounced; sensory disturbances, vasomotor perversions and symptoms of visceral crises and muscular atrophy are most characteristic.

Diagnosis.—This disease may be recognized by a careful observation of the symptoms previously mentioned. There are several differentiations to make. The characteristic rigidity and the deformities of *amyotrophic lateral sclerosis,* together with the absence of marked anesthesia and sphincter involvement should help us to exclude this disease. In *progressive muscular atrophy,* the muscular wasting starting in the thenar and hypothenar groups, together with the systematic atrophy of other muscles preceding the paralysis, should define that disease. The *ataxia* of *tabes* should not make that differentiation difficult. In *pachymeningitis* the positive history of injury, the pain, the lack of sphincter implication and the tendency to a cervical lesion should aid us in deciding. *Tumors of the cord* are painful, develop slowly, and represent a defined localization. *Brain palsies* are generally unilateral, painless and not so spastic.

Prognosis.—The outcome is always unfavorable. The disease either remains in its chronic condition or secondary degeneration may follow. While improvement may at times be noticed, the tendency is toward a permanent paralysis. The cause and the location of the disease have much to do with the prognosis.

Treatment.—Measures of all kinds have been resorted to with the aim of giving local relief. It is a question if these are not merely suggestions to the patient. The counter-irritant, the cautery and the seton have never obtained results. Galvanic, faradic and static electricity have been adjuvants of limited power. Hydrotherapy, mechanical and surgical measures offer assistance only in surgical cases and as general adjuvants. The treatment depends entirely upon the individual demands. No stereotyped applications should be depended upon. Everything which looks to the relief of suffering or the suppression of the pathologic process should be employed, but no specific cure exists.

Among the remedies indicated we may consider *arsenic, mercurius, phosphorus, potassium iodid* and *aurum chlorid.* The remedies for the acute form have already been mentioned. *Arsenic* applies most decidedly to the conditions of disease produced by tissue degeneration; it refers to the patient who shows the result of anemia or malnutrition; exhaustion and restlessness are its characteristic symptoms; a dry skin, with much itching and burning, is always a feature. Furthermore, this remedy relates to a weakness in the back attended by pain, heaviness and some spasticity of the limbs; it is, therefore, frequently indicated in this disease. In order to obtain the best results, it should be given interruptedly for a long time. *Mercurius* frequently points to a toxemia from drug or specific infection; usually the glands are involved primarily and the necrosis affects all the tissues later; the pains and weakness of the limbs are attended by a cold clammy sweat or other signs of sepsis. *Phosphorus* is always thought of when fatty degeneration or phlebitis are pathologic conditions; numbness is generally associated with the muscular weakness of the limbs; a pseudo-muscular hypertrophy is a possible condition as the disease progresses.

Potassium iodid is to be considered when the disease is secondary to syphilis or other dyscrasia; the pains, in this case, are deep-seated, contractions are frequent and the exacerbations are mostly at night; glandular induration and necrosis of the skin are often in evidence. In this respect a comparison should be made with sulphur, lachesis, silicea and calcarea fluorica. *Chlorid of gold* and *argentum nitricum* are remedies with similar indications, but they are related more particularly to the sclerotic condition of nerve tissue. *Plumbum* should be thought of in connection with these symptoms when there is also a tendency to glandular induration and ulceration. *Conium* has many features which answer for the chronic stage of myelitis; the trembling and painful stiffness of the limbs, the sphincter inability and the symptoms which follow from sexual weakness are most illustrative. *Mezereum* may be prescribed when neuralgias, bone pains and subsultus are associated with a general bruised sensation in the limbs. With this we may compare *arnica, rhus tox, phytolacca and mercurius.* Other remedies such as *zinc pic., zinc val., cuprum met., agaricin, atropin* and the *iodid of arsenic* may be applied in certain cases. *Strychnia,* which has been used so much in the past, is now generally discarded unless cardiac insufficiency is present.

ACUTE ANTERIOR POLIOMYELITIS.

This disease is an acute myelitis involving the anterior horns of the spinal gray matter. It is marked by a sudden onset, pronounced fever and a paralysis which is followed by atrophy of the muscles involved. It has no sensory phenomena and is most liable to occur in children.

Etiology.—The majority of cases are found in children between six months and two years of age. Traumatism, colds, exposure and dentition are claimed as causes. Infectious fevers probably are the most common cause. Malnutrition, occurring at a time when the spinal cord activities are being developed, no doubt is an underlying condition which favors it.

Pathology.—The pathology is that of a localized myelitis involving the multipolar ganglion-cells of the anterior cornu, with the characteristic inflammation, necrosis and even cavity-formation. Finally sclerotic changes in the affected area and even in the pyramidal tract with atrophy of the anterior nerve-roots and the muscles thus supplied may be observed. Fatty degeneration of the muscles is not uncommon.

Symptoms.—Without prodromal signs or an apparent cause the patient experiences moderate fever (100°-102° F.) and this may be attended by convulsions, vomiting and other visceral disturbances. Soon after, a few hours or a few days, paralysis and flaccidity of one or more limbs is observed. If the paralysis develops very slowly, it is termed a subacute form. As a general thing the prodromal symptoms are not marked, sensations are normal and the paralysis, which most frequently appears suddenly, is often the first symptom observed.

A *stationary period* exists in the paralysis after it reaches its height, which may continue several weeks. Wasting of the paralyzed limb may then be noticed, the muscles become flabby, the reflexes are absent and tenderness is pronounced. A stage of improvement now follows and this may continue for six months or a year. During this time the paralysis gradually disappears, improvement beginning in the parts least affected. This may continue until one limb or only certain muscles are affected. The period of improvement depends decidedly upon the physical condition of the patient and the extent of the paralysis. The case will finally lapse into a chronic stage which may continue for the remainder of the patient's life.

The *lower extremities* are involved more frequently than the upper, and the left leg more than the right, while a crossed form, affecting one upper with the opposite lower limb is not rare. In the upper extremity the muscles of the hand, the extensors of the wrist and the deltoid are chiefly involved, while in the lower limbs the extensors are mostly affected. The *atrophy* develops from the inaction of the muscles which receive nerve fibers from the affected cells. The atrophy continues until fibrous degeneration occurs, which permits contractures of the muscles. Luxations and other deformities are, therefore, natural sequelae of prolonged cases of this disease. The arm may not only atrophy, but it is liable to dislocation. On account of the involvement of the extensors of the lower extremities foot drop, talipes equinus and flexures are common.

Diagnosis.—During the early stages of acute poliomyelitis it is possible to mistake the symptoms for acute rheumatic fever. When the paralysis is established the diagnosis should not be mistaken, especially with the appearance of the different stages. *Cerebral palsies* of children have initial convulsions affecting definite muscles. *Multiple neuritis* has marked sensory disturbances while *progressive muscular atrophy* shows the characteristic atrophy which precedes the paralysis. *Myelitis* gives slight febrile warning, the paralysis appears suddenly, involving all the muscles below the lesion, sensory disturbances are always present, while sphincter involvement is invariably present. In addition to this we may say that faradic reaction is lost earlier in anterior poliomyelitis than in other simulating diseases.

Prognosis.—A fatal termination in this disease is rare. Improvement generally takes place in the majority of cases after the stationary period. Complete recovery, however, is not usual. If the atrophy is pronounced, the paralysis will continue. Early and persistent treatment will accomplish much in preventing extreme and permanent disability. Very frequently the disease shows signs of recovery in all except perhaps one limb.

Treatment.—In the first stage our treatment should be directed to the consideration of the febrile condition. The patient should be put to bed and kept absolutely quiet. The tepid sponge and a high enema should be given, and any applications necessary for the relief of local symptoms should be resorted to. The affected limbs should be kept warm, if necessary, by means of cotton and the roller bandage. *Aconite* is the most frequently indicated remedy in this stage. *Belladonna, chamomilla, pulsatilla, eupatorium, gelsemium, hellebore, baptisia* or *rhus tox* may be demanded.

In a few weeks, when the acute or febrile symptoms subside, it is advisable to begin the use of electricity; this should not be used too frequently at first. It is often best to suspend this treatment after a time and give the affected limbs a more complete rest. The merits of galvanism and faradism depend upon the character of the palsy. When the paralysis is complete, the galvanic current seems to give the best stimulation. The *electricity* should never be given long enough or strong enough to exhaust or tire the muscles. *Massage,* friction and the alternate douching of the parts with hot and cold water will add much toward restoring the lost muscular tonicity. Inunctions of oils and fats will aid the trophic rejuvenation.

Later *passive motion* should be systematically practiced to overcome the tendency to spasticity or muscular contractions. The limbs should not be allowed to remain too long in a pendant position, and they should be kept warm at all times. *Exercise* should later be instituted to develop the normal muscular activity, which has been suspended by the different stages of the paralysis. The different forms of physical exercise, gymnastics and bicycle riding will often encourage improvement in the activity of affected muscles.

In *the chronic stage* such remedies as *plumbum, picric acid, graphites, causticum, alumina, mercurius, platinum* and *zinc* will be indicated.

ACUTE AND CHRONIC SENILE PARAPLEGIA.

The cause of the *acute variety* of this disease is found in a thrombosis or hemorrhage of the spinal arteries. The paraplegia develops suddenly, as in acute myelitis, only the pain is not so decided. The paralysis varies considerably; often it is complete and the symptoms seem to point to myelitis; again it may be only partial and there are signs of intermittent improvement from the very beginning. The serious phase of the pathologic condition depends upon the arteries involved; if the central are affected, the condition is more serious. *Ataxia* seems to be a decided symptom in these cases. Improvement appears early, though relapses are liable to occur. The remedies mentioned under myelitis will generally be indicated for this disease. Rest and nourishment are essential considerations.

The *chronic form* of senile paraplegia begins with a simple weakness of the legs; the muscles affected gradually atrophy and the paraplegia develops slowly. The arms are involved later in the disease; the sphincters are slightly paretic. The similarity to *progressive muscular atrophy* is evident, but the paralysis and wasting develop together, the legs are generally involved first and there are no fibrillary contractions. Chronic progressive *anterior poliomyelitis* is the most nearly allied to this. The treatment would not be different from that of poliomyelitis in the later stages. The outlook depends upon the age and strength of the patient, and the condition of the blood vessels.

LANDRY'S PARALYSIS.

Landry's disease, as understood today, is an acute infectious ascending paralysis. The toxemic conditions which produce it are confined mostly to the anterior gray matter of the cord. The spinal roots, the medulla and cortex are often involved by the progressive pathologic process. The paralysis begins generally in the legs, extends to the trunk and arms and quite rapidly ascends to the medulla. The respiratory and throat muscles are generally involved, and hence sudden death is usual. The sensory disturbances are not marked, the sphincters are rarely affected, the faradic irritability is normal and muscular atrophy is rare.

Etiology.—The disease occurs most frequently with men between the ages of twenty and forty, but it is exceedingly rare. *Exposure* may be an exciting cause, though it often appears without any known cause. *Infection,* without doubt, is the principal factor. *Pneumonia,* typhoid, diphtheria, syphilis, smallpox, tuberculosis and influenza are looked upon as predisposing diseases. The infection evidently reaches the gray matter through the arteries which approach the anterior cornu along the motor roots and this explains the limitations of the myelitic process.

Pathology.—A variety of diseases have been confused with Landry's paralysis, but it is now quite thoroughly settled that it is due to a poison of microbic origin, the toxemia affecting the gray matter of the anterior horns and their motor roots. The cerebral gray matter has been involved in a few cases. A *diffuse myelitis,* or a general neuritis, are possible sequelae when the toxemia is extreme. A form of rabies known as "paralytic rabies" causes a disease similar to this. The consensus of opinion favors the theory of its being an acute polyneuritis.

Symptoms.—*Premonitory symptoms* are rare, though there may be slight numbness in the extremities, with some pain in the back and limbs. Usually the first symptoms are observed during or after an infectious disease. In a few days the lower extremities are completely paralyzed. Respiration and deglutition are perverted, and in severe cases every muscle below the face is paralyzed. The cranial nuclei may be involved and the oculo-motor, the facial and the hypoglossal centers being most frequently affected. Following the invasion of the respiratory center, we may observe pronounced mental perversions and cardiac failure. The disease is, therefore, rapid in its development and a fatal termination is usual, although some recoveries are reported.

Pain and *sensory disturbances* are not marked and the reflexes are only slightly disturbed. The sphincters are not affected and as the trophic functions are not impaired, there is little muscular atrophy. As the disease progresses so rapidly toward the medulla, it generally terminates fatally in less than a week. If the medulla is not reached, the patient may be completely paralyzed below the head. Under such conditions as this a slight improvement may take place or the paralysis may remain stationary for a time and possibly recede in the reverse order of its appearance. In some cases the upper extremities may show the first symptoms.

Prognosis.—Nearly every case is liable to terminate fatally after a short duration. At best the patient is liable to remain a hopeless paralytic.

Diagnosis.—With the above symptoms a diagnosis is not difficult. Acute myelitis, acute poliomyelitis and acute multiple neuritis must be differentiated. The acute ascending course, the absence of fever, anesthesia, atrophy, sphincter troubles and degenerative reactions should help us to decide positively.

Treatment.—Little may be hoped for from any treatment. The local applications and remedies mentioned under myelitis should be considered.

TABES DORSALIS.

Locomotor ataxia is a common disease of the spinal cord which represents eventually a progressive sclerosis of the posterior fiber tracts. It may begin in the posterior spinal ganglia or the peripheral sensory nerves, and it is characterized by incoördination, Argyll-Robertson pupil, severe pains, loss of knee jerk, anesthesia and many visceral and trophic disturbances. It may assume various forms which have given rise to many different descriptive terms. The typical form, however, will be considered in this chapter.

Etiology.—Middle life seems to be the average age for its appearance, though the marked symptoms may not appear until a later age. If heredity is a cause, it may be observed in younger patients. *Syphilis* is the most frequent cause, though it is claimed that other toxic factors may produce a similar degenerative condition, the larger percentage may be traced, directly or indirectly, to syphilis. *Exposure,* extreme and persistent muscular exertion, sexual excess and unnatural functional activities may produce a similar set of symptoms, but these do not characterize the typical disease. Syphilis seems to predispose the nerve fibres to the degenerative process observed in tabes. It is not unreasonable to believe that the lack of thorough antisyphilitic treatment is responsible for its frequent development of tabes. The *male sex* is more frequently affected. The *neurotic inheritance,* hysteria, chorea, hemiplegia and epilepsy may have something to do with its development. *Acute infections,* such as typhoid, pneumonia and rheumatism, severe hemorrhages and even diabetes have been claimed as influencing factors.

Pathology.—As Dana says: "We can not understand the pathology of locomotor ataxia without acknowledgment of the pathology of syphilis in its relation to the nervous system." He speaks of tabes "as a post-infective degeneration, which first attacks the posterior spinal ganglia or corresponding cells of the special senses, due to a prolonged poisoning of these parts by the toxins of the infection." The syphilitic infection seems to make its first impression on the posterior roots because the veins and lymphatics are unable to eliminate it. Later it involves a vertical section between the columns of Goll and Burdach and gradually these columns suffer from the sclerotic degeneration which begins its development toward the medulla. The *pathologic changes* are first inflammatory, showing the effect upon the blood vessels and serous membranes by an exudate. The degenerative changes in the nerve fibers follow directly and this continues until connective and neuroglia tissue supplant the sensory or motor tracts as the disease progresses. The pia mater and the walls of the blood vessels also present a slight thickening.

In *appearance* the cord shows a reduction in size and a slight anteroposterior compression; the posterior columns are contracted wherever the disease is established. The segments involved most in the beginning are the upper lumbar and lower dorsal, while the column of Lissauer also shows early degeneration. As the columns of Goll and Burdach are transformed into a mass of hardened connective tissue, portions of the lateral cord are liable to be implicated, though not extensively. The gray matter of the posterior cornu may show some signs of degenerative change and the cells are decidely atrophied in advanced cases. The anterior roots are not generally involved in the pathologic process.

Sometimes the *initial lesion* starts as low as the sacral segment, and in such cases we notice the marked genito-urinary disturbances. When the *cardinal symptoms* of tabes are present, we generally locate the lesion in the upper lumbar or lower dorsal region. There is a rare form known as the brachial or arm type in which the pathologic change develops first in the cervical region. The peripheral nerves, and the cranial nerves, may be affected during some stage of the disease. *Optic atrophy* may appear early and progress with the disease. The third, fifth and sixth nerves are occasionally involved. The vagus and glossopharyngeal often show a sclerotic tendency in the early part of the disease.

Symptoms.—There are three stages in the symptoms: the initial or pre-ataxic, the ataxic and the paralytic. In *the initial stage* there is a slight disturbance of the muscular sense; the patient can not walk well without the aid of vision; the incoördinate condition is noticed when walking in the dark, and stumbling occurs at the slightest obstruction in his way. The limbs are numb and heavy; the ground does not feel natural under his feet and there is a general sense of insecurity when he walks. The sexual power is weakened and the bladder function is imperfect. The knee reflex is lessened or lost and there is a tendency to exhaustion of the limbs from the slightest exertion. Numbness and slight anesthesia are detected in the feet and the lower part of the legs. These symptoms may persist for several months or even years.

The *ataxic stage* is marked by the increased incoördination. The patient is very unsteady while walking. He watches his feet and spreads the legs at every step, depending upon the use of a cane for support. Romberg's sign (inability to stand with the feet close together and the eyes closed) is present. He can not walk backward when the eyes are closed; it is impossible to turn about quickly or to rise from a sitting position at command. There is a tendency to rigidity of the advancing leg, the supporting knee is over-extended and the heel strikes heavily upon the floor. In attempting to sit down suddenly the knees give way and he drops into the chair; in crossing the knees the limb is raised too high and drops quickly. All of these symptoms show the increased loss of the muscular sense and the development of ataxia.

The *sensory symptoms* are even more pronounced. The feet feel as if they were encased in cotton; fulgurating or lightning-like pains attack the legs, and anesthesia is manifested by delayed sensations; sometimes a girdle sensation is felt in the back. The sexual power is now absolutely lost, and the bladder and rectal sphincters are paretic. The pupils are small and stiff, reacting to accommodation, but not to light (Argyll-Robertson pupil). Symptoms of visceral disturbance may begin at this time.

The paralytic stage is reached in a few years unless the pathologic process is arrested. Some cases may show signs of early improvement while the second stage may be prolonged for years without paralytic development. Therefore, persistent and careful treatment may do something for this incurable affliction. The word paralysis is hardly a proper term, inasmuch as the condition is an extreme degree of ataxia, which is practically a paralysis. In this condition the patient loses the entire power of locomotion. *Muscular atrophy* now appears in the unused limbs; *anesthesia* is complete, the bladder is paralyzed and the rectal sphincter incontinent. The extreme pains subside somewhat, but the patient suffers from pronounced exhaustion. During this stage there may be some involvement of the upper extremities, but this is generally slight. The patient may linger now for some time as a hopeless paralytic.

There are other attendant symptoms of tabes which demand some consideration. For instance, we find *neuralgias* associated with the fulgurating pains, the latter appearing generally in paroxysms and extending down the legs often causing involuntary contractions. The neuralgias are located mostly in the bladder and rectum and may cause excruciating suffering and functional derangement. These pains are the most distressing symptoms of the disease. *Hyperalgesic areas* are also observed in various parts of the body, which may be the foci of the lightning-like pains. *Analgesia* is also common and may affect the deeper tissues as well as the skin, the patient often not observing this until the test is made by the prick of a pin. This analgesia is not confined to the limbs, generally occurs in plaques and is usually symmetrically distributed. *Retardation* in the transmission of sensations increases slowly as the disease progresses. The senses of touch, temperature and pain are often affected differently. Pain may be retarded while touch is acute, and again there may be absolute loss of temperature-sense with only slight change in touch and pain-sense. The tactile sensations are preserved longest. *Sudden involuntary jerkings* of the fingers, wrist, arm or foot may appear independently or in association with the fulgurating pains. These simulate athetoid movements, but are not the same in character.

The *ocular muscles* are often implicated, especially those supplied by the third and sixth nerves, though other ocular paralyses may be observed. All of these are liable to be transitory, the permanent palsies appearing later. The early palsies are usually due to gummas which may disappear under treatment, while the later palsies, in contra-distinction, represent a degeneration of the nerve nuclei and their surrounding tissues. *Myosis* may occur when the dilating sympathetic fibers are involved. The loss of light reflex and pupillary rigidity are defined under the term *Argyll-Robertson pupil;* the failure of the pupil to respond to light appears early and later we may observe the loss of accommodation. The pupils may also be irregular, while ptosis is a common symptom. *Optic atrophy* is found in about 10 per cent of the cases. It usually begins in the early stage. The atrophy is generally bilateral, though it may affect one eye more than the other. At first a contraction of the color field is detected, next the form field shrinks, and lastly the vision is diminished or lost. Hemianopsia and central scotoma are possibilities.

The *auditory nerve* is also liable to suffer degeneration in tabes, but this is more frequently related to middle ear disease. The impairment of hearing develops slowly and is often bilateral. Tinnitus and aural vertigo are frequently recognized. The senses of smell and taste are slightly perverted in some cases.

Visceral disorders may be the most disturbing features of the disease, manifested by attacks of pain and sudden functional derangement of the stomach, intestine or bladder. *Gastric crises* are the most common. Pain may suddenly appear in the gastric region and radiate to the abdomen, chest and back; the paroxysm is insufferable and symptoms resembling those of angina pectoris may be present. Vomiting may be violent and persistent. While it is not always excited by the ingestion of food, the stomach is rarely able to retain nourishment during these paroxysms. Diarrhea, extreme exhaustion and collapse are not infrequent. These attacks may terminate as abruptly as they begin. *Intestinal crises* may occur independently of the gastric crises, and are characterized by diarrhea or constipation, colic or rectal tenesmus. *Urinary disturbances* may arise in the early stages and is recognized by the sphincter impairment. Paroxysmal pain, however, may appear suddenly during the progress of the disease; complete retention of urine and cystitis are then very troublesome. Glycosuria may also be discovered in tabes. The urea, phosphates and chlorids may be diminished and the amount of urine may be increased or decreased. The inflammation along the urinary tract may be so great that *renal crises* may be observed. The generative function is always modified in this disease. Impotence appears early, though sexual excitement may exist for a time.

Pharyngeal and *laryngeal disturbances* have been noticed in many cases; pharyngeal crises are observed by the repeated, painful and noisy effort at swallowing; laryngeal crises are recognized by croupy attacks with a tendency to dyspnea and cyanosis. *Bronchial crises* are manifested by unexpected and violent fits of coughing, while *cardiac crises* simulate an attack of angina. Arteriosclerosis and organic heart disease often attend tabes.

Trophic perversions in tabes are characterized by the general malnutrition. A round perforating ulcer of the sole of the foot is significant of the later stages of tabes. Herpes, eczema, urticaria, pemphigus and ichthyosis are sometimes sequelae. When syphilis has not received proper treatment, we may find incurable ulceration of the throat, tongue or mouth, and the teeth may fall out.

The *arthropathies* which attend the pronounced cases of tabes were first described by Charcot. These are characterized by a sudden painless and enormous swelling of the joints and adjoining structures, which may rapidly involve the whole limb and usually subside after a short duration. *Spontaneous fractures* and *luxations* varying in degree commonly occur when the trophic condition has reached this stage.

Diagnosis.—This is difficult only in the early stages. Many cases in the pre-ataxic stage are erroneously treated for rheumatism or neuralgia. The loss of knee-reflex, the presence of fulgurating pains, the Argyll-Robertson pupil and Romberg's sign leave no doubt as to the disease. The otherwise unexplainable absent knee-jerk with any other of the characteristic symptoms, should lead us to suspect the presence of tabes. In *hereditary ataxia* the peculiar pains are absent, the hypoglossal nucleus is involved early and the disease generally begins in childhood. *Multiple neuritis,* chronic myelitis and spinal tumor are differentiated by their characteristic symptoms. *Paraplegias* are marked by exaggerated reflexes, ankle clonus and definite areas of dysesthesia. *Cerebellar disease* is characterized by optic neuritis, severe headache and vomiting and a different form of incoördination. The knee-reflex is usually preserved.

Prognosis.—Complete recovery, in a typical case, is not to be expected. Some relief of symptoms may be obtained in the early stages.

Treatment.—It is possible, by the use of local measures and internal remedies, to limit to a certain degree the extent of the pathologic development. If the case comes under our care in the pre-ataxic stage, we may possibly relieve the loss of muscular sense and stop the progress toward complete incoördination. While the degenerative progress may be checked it is impossible to regenerate the tissues involved. Much depends upon the early management of the syphilitic invasion. Relief from mental and physical strain, and often a change of vocation or climate may temporarily benefit some cases.

Any contributing autotoxemia should be removed by attention to the elimination by bowels, kidneys, lungs and skin.

Absolute rest in bed may lessen the nerve exhaustion. The *diet* should be highly nutritious. *Hydrotherapy,* in the nature of warm baths or possibly the hot or cool spinal spray, are of some service, especially early in the disease. *Electricity,* both faradic and galvanic, has been assiduously employed, but the results have been negative. The psychical effect upon the patient is, however, very pronounced. In this respect the static spark, no doubt, yields the best results. The actual cautery used on the spine, cupping and blistering have never given any satisfactory results. *Suspension* has perhaps given some relief to the spinal irritation, but no decided benefit has been obtained by this. Fränkel's system of motor education applied to the affected muscles has to some extent overcome the extreme ataxia. The fulgurating pains often demand palliation. *Morphin* hypodermatically, is probably the best agent for this purpose. *Codein* or *heroin* are also valuable. The *coal-tar derivatives,* harmful as they may be in some cases, are often used.

If any relief of symptoms is to be obtained at all it must come from *internal medication.* In this respect it is natural to look back to the creative cause. The Practical Medical Series, September, 1904, however, says, "the number of cases in which antisyphilitic therapy has caused an aggravation of the symptoms is much greater than the number helped by such treatment. It would, therefore, seem that antisyphilitic treatment in tabes should not justify the confidence lately given it." If we employ antisyphilitic treatment the *iodid of potash* should be given in ten drop doses and gradually increased. *Argentum nitricum* is a serviceable remedy and should be used in the third decimal potency, freshly prepared, for a considerable time. This remedy refers to the tremulous weakness, the paralytic heaviness and sensory disturbances in the legs. Some good results have been claimed from its use in quarter-grain doses three times daily, but the probability of permanent skin discoloration should be kept in mind. In line with this we should consider the *chlorid* of *gold* in the second decimal potency.

Zinc is another remedy which is useful in all degenerations of the nervous system; it may be employed in this disease in various forms. Where there is general nervous exhaustion the *phosphid of zinc* is best; for the fulgurating pains and other sensory disturbances, the *picrate of zinc* should be used; when general nervous excitement exists, the *valerianate of zinc* will be indicated. In anemic cases *ferrum picricum* or *ferrum phos.,* may be employed. *Alumina* has been recommended when dizziness, exaggerated sensations and the characteristic constipation exists. *Phosphorus, phos. acid* and *nitric acid* are also worthy of consideration. The symptomatology of *secale* points more clearly to the genuine features of tabes than any other remedy. Considerable praise has been given to this when given in potency. A careful study of its action in many clinical cases has satisfied me that no appreciable results are obtained.

Causticum has been recognized by many as a valuable remedy in nervous diseases, but its sphere of action, however, is no doubt limited; where there is muscular tension and arthritic pains it may be serviceable; its action upon the sphincters is pronounced and for that reason it may give some favorable results in the early stages. *Cuprum metallicum, gelsemium, cannabis sativa* and *esculus* may be considered in this respect. The *iodid of mercury* and the *iodid of arsenic* should not be overlooked in considering the syphilitic cause. *Colchicum, ignatia* and *physostigma* may sometimes be indicated. *Cicuta virosa* (tr. m. 5) four times daily, may give relief when cerebrospinal irritation, with tetanic and epileptiform symptoms are present. *Cimicifuga* may be used with good effect when rheumatic pains or stiffness are present. In fact we may safely say that any remedy indicated for any set of peculiar symptoms may be used in this as well as any other disease.

ATAXIC PARAPLEGIA.

This is a form of paraplegia which is ataxic and yet spastic in character and represents a combined posterior and lateral sclerosis.

Etiology.—The process is a secondary cord-degeneration. A diffuse myelitis which invades the lateral tracts may be added to a primary tabetic condition and as a result of this, ascending and descending sclerosis may occur. General paresis, leptomeningitis and vascular lesions may also be the initial causes. Syphilis is less rarely a factor than traumatism and exposure.

Pathology.—Both the posterior and lateral columns are invaded. The sclerosis in the columns of Goll begins in the middle and lower dorsal regions and extends upward to the medulla. Burdach's column is affected only slightly. The pyramidal tract is involved mostly in the outer area, while the direct pyramidal tract is involved only slightly in the upper cervical region. The direct cerebellar tract is extensively sclerosed. Thus we find the posterior columns undergo an ascending sclerosis and the lateral a descending sclerosis.

Symptoms.—The symptoms are those of spastic paraplegia and locomotor ataxia combined. Thus we see a similarity in the symptoms of chronic myelitis and tabes dorsalis which may vary in different cases. There is incoördination with an ataxic gait; anesthesia, paresthesia and fulgurating pains are invariably present, though the muscles are rigid, the tendon reflexes are exaggerated and ankle clonus is obtained. In the early stages the symptoms strongly simulate ataxia, but the exaggerated reflexes and the peculiar rigid posture and gait suggest at once a different disease. Muscular weakness is pronounced, but the sensory symptoms are not so marked as in typical ataxia. Visceral crises are uncommon and, if present, they are light in character. The upper extremities also show a muscular spasticity. Clonic spasms involving the muscles of the lower jaw may be frequently demonstrated. The pupils may be irregular and sluggish, but the typical Argyll-Robertson pupil is not usually present.

Diagnosis.—The differentiation considers the symptoms of tabes and those of chronic myelitis. In the early stages ataxia is prominent. Later, as the paraplegia develops, the muscular weakness, exaggerated reflexes and the absence of decided sensory and visceral symptoms should settle the diagnosis. *Cerebellar tumor* may give some of these symptoms, but the occipital pain, choked discs, retracted head, the vomiting and mental symptoms, indicate the brain involvement. It may also cause forced attitudes or movements.

Prognosis.—The danger to life does not seem to be so decided as in tabes. There is even more hope of improvement, though a complete cure is not to be expected. Its course of development is not so rapid as that of tabes.

Treatment.—The general and remedial treatment has been suggested under chronic myelitis and tabes. The local use of massage, electricity and hot or cold spinal douches, may relieve the muscular spasticity. *Valerianate of zinc, agaricin, hyoscyamus, hydrobromate of hyoscin, gelsemium,* etc., may be indicated.

THE COMBINED SCLEROSES IN ANEMIAS AND CACHEXIAS.

Pernicious anemia and the cachectic conditions, according to the observations of Putnam and Dana, may create degenerations in the posterior columns and the pyramidal tracts. Associated with this pathology we may also find a slight disintegration of the nerve cells in the gray horns. The toxemia is incident to a systemic debility and affects the posterior sensory fibers and the lateral descending tract. Hemorrhagic foci appearing in these areas give rise later to a sclerotic transformation.

Etiology.—The disease is observed most frequently in women beyond middle age. Malaria is the most common cause, though any prolonged toxemia may create it. Diseases of the suprarenal glands, heredity and neuropathic conditions are also considered as predisposing factors.

Symptoms.—The signs of anemia precede and attend the characteristic symptoms. Emaciation, diarrhea, coldness of the extremities and general exhaustion are present early. Numbness of the extremities generally precedes the ataxic paraplegia which is the typical symptom. The spasticity, exaggerated knee-jerk and ankle-clonus appear early, but may subside somewhat if the cause of the disease is removed. Paresthesia, anesthesia and neuralgia may be present in the later stages. The vision and other special senses are not affected, but there may be slight visceral disturbances.

Diagnosis.—The presence of anemia or cachectic diseases, the age of the patient, the slight ataxic paraplegia, the emaciation and progressive weakness, with the paresthesia, will settle the diagnosis.

Prognosis.—There is more hope for a cure in this disease than in any of the ataxias. Pernicious anemia, however, does not respond readily to treatment. The prognosis depends upon the extent of the causative disease and the degree of cord involvement. The *treatment* should be applied to the underlying causes.

FRIEDREICH'S ATAXIA.

Friedreich first called attention to the fact that there are certain forms of ataxia which may be properly termed family, or hereditary ataxia. Some of these are purely congenital, while others develop so early in life that the hereditary feature is clearly established. The spinal ataxia which is defined as Friedreich's, closely resembles hereditary cerebellar ataxia and hereditary ataxic paraplegia. The cerebellar form shows a pathologic limitation to the cerebellum, the hereditary ataxic paraplegia develops mainly in the lateral columns, but Friedreich's is an ataxia due to a chronic degeneration in the posterior and lateral columns of the cord.

Etiology.—A neuropathic history may be obtained in nearly every case. Males are most frequently affected, though the transmission is by the female line. Usually the history of insanity or inebriety is found in the preceding generations. Syphilis in the parents is a frequent though not invariable cause. Tuberculosis and consanguinity may be predisposing factors. It seems to appear most often in large families. The degeneration of the posterior and pyramidal columns seems to occur at the time of cord development, when malnutrition or hereditary dyscrasia would disturb it most.

In some cases the disease is congenital, in others it develops early in life or at the time of puberty and adolescence. The first or second dentition, and particularly infectious diseases, may be exciting causes. Locomotor ataxia in parents does not predispose to it.

Pathology.—There is marked sclerosis of the columns of Goll and Burdach and the same degeneration exists in the pyramidal tracts. These are the parts which are always involved and in typical cases the sclerosis is complete. Gower's tract and the direct cerebellar tract may be slightly affected in certain cases; the anterior or direct pyramidal tract may also show some involvement. The posterior horn shows little disturbance, though the cells may be somewhat shrunken. The cord is small in size from the congenital defect in development. This sclerotic condition is also found in the medulla and particularly about the hypoglossal nucleus. The posterior spinal roots and, to a certain extent, the anterior roots, suffer a similar degeneration, while the peripheral nerves are still less involved.

Symptoms.—If the disease is congenital we notice that the child does not use the tongue well. The mind develops very slowly and there is inability to walk at the proper period, while later, deformities of the legs appear. In other cases clumsiness in walking is first observed; the child stumbles easily and there is a general unsteadiness on the feet, and finally ataxia is pronounced. Previous to this there are few sensory symptoms which suggest its development, though numbness and slight pain may exist. The knee reflex is lost, but the muscle sense is not greatly affected. Coarse and rhythmic movements in the legs occur in attempting to move them. The same tremor is noticed in the facial muscles when there is an attempt to speak.

In the *ordinary case* in a few years the arms are affected and then the real *bulbar paralysis* appears. The *speech* is thick or scanning until the tongue refuses to work. *Vertigo,* nystagmus and severe headache may develop at this time. A peculiar *mental dulness* with an expressionless facial appearance may be observed. The patient is apathetic, and oscillation of the head and choreic symptoms are common. After a time the legs become weak, there is some atrophy and paraplegia finally appears. Upon examination of the spine a *lateral curvature* may be detected. Later *club foot* is liable to develop. The *vesical* and rectal sphincters are rarely affected. Visceral disturbances are not evident in the average case.

Diagnosis.—The age, the character of the ataxia, with upper arm and bulbar symptoms, the normal condition of the muscular sense, the lack of sensory symptoms, the infrequency of optic atrophy, the scoliosis and club foot ought to decide the diagnosis. In addition to this the atypical ataxia and the family tendency should help us. In *tabes dorsalis* we have rarely a cerebellar involvement, and besides there are characteristic visceral crises, absent knee-jerk, lightning-like pains and other sensory perversions, pupillary rigidity and vesical weakness. *Chorea* may resemble it in the early stages, but this should be distinguished by its peculiar twitchings and the absence of ataxia, bulbar symptoms, scoliosis, club foot, etc.

Prognosis.—The outlook is always unfavorable. The patient may linger for years, but the ataxia eventually reaches the incurable or paralytic stage.

Treatment.—Much may be done in the way of overcoming malnutrition. In this respect a nutritious diet, outdoor life as much as possible, with massage and inunctions of oil may avail somewhat. Under this head also we should consider the internal use of *calcarea carb.* and *calcarea phos., calcarea iodid, arsenicum* and *silicea, ferrum phos.* and *ferrum iodid, calcarea fluorica, sulphur* and that class of remedies. The scoliosis may be helped by a brace. Careful education of the mind and muscles may relieve the distressing symptoms, but other than this we can do little.

HEREDITARY SPASTIC PARAPLEGIA.

This is a disease which is paraplegic in character and yet has many ataxic symptoms. It appears in early or middle life and is hereditary, though it may follow infectious diseases. Syphilis is not a cause. The pathology is marked by degeneration of Goll's column, the pyramidal tract and slight portions of the direct cerebellar tract. Stiffness and leg weakness are early symptoms; there is an imperfect use of the leg muscles which gives the appearance of ataxia. The reflexes are exaggerated and ankle-clonus is present. Sensory disturbances and sphincter weakness are not recognized. The arms and possibly the cranial nerves may be implicated later.

The disease is not common, its course is not rapid and, though a perfect cure is generally impossible, the danger to life is not pronounced. The general management is similar to that of other ataxic conditions; the remedies should be given in accordance with the demand of symptoms.

MULTIPLE CEREBRO-SPINAL SCLEROSIS.

This disease has also been termed multiple sclerosis, insular sclerosis and disseminated sclerosis. It is pathologically characterized by the development of sclerotic patches irregularly distributed in the brain and cord. These changes are primarily vascular and are probably due to some infection or intoxication. It is clinically characterized by some paralysis, usually paraplegia, intention tremors, scanning speech, nystagmus, mental symptoms and other disturbances depending upon the location of the sclerotic patches.

Etiology.—The disease is not common and it affects both sexes alike. In rare instances children are affected. *Heredity* apparently has little influence, though the neuropathic tendency is discovered in many cases. *Traumatism,* exposure and excesses probably have little to do with its development. The *infections* are at present regarded as the chief etiologic factors. Intoxicants are still regarded as causes by many experienced observers. Whatever the cause, it is evident that the "irritative influence" reaches the nerve tissue involved, directly through the blood channels.

Pathology.—*Macroscopically* the sclerotic patches are of various sizes and are usually grayish or pinkish-gray in color, The white substance is invaded most frequently though the gray matter does not escape. There is no uniformity in the sclerotic distribution, the cranial nerves, their nuclei and the spinal roots often being invaded. *Microscopically* the patches are distinguished from the surrounding normal tissue by their definite outlines. The nerve fibers included in the sclerotic area are deprived of their myelin, though the axis cylinders show slight change. The proliferation of neuroglia tissue is always apparent, but secondary degenerations are not generally found. Popoff, however, denies the neuroglia hyperplasia, claiming that the change is due to the degeneration of myelin and axis cylinders. The nerve cells are decreased in size and often pigmented. The blood vessels at the seat of the lesion are decidedly changed, their coats, especially the external, being greatly thickened. "The perivascular sheaths are dilated and at times obliterated." Thrombotic and embolic changes are also occasionally found.

Symptoms.—The *motor symptoms* are marked. Aside from the muscular weakness the limbs are decidedly rigid, causing a characteristic spastic gait which is often combined with cerebellar staggering, thus giving rise to the term "cerebello-spasmodic gait." The reflexes, except in the early stages, are exaggerated and clonus is usually present. The upper extremities display the same spastic tendency with some rigidity. The *intention tremor* is a classical sign and this is evidenced when the patient attempts to take something with the hands. It is coarse in character and the trembling may affect all muscles which are called into use for a particular motion. When the parts are at rest the tremor disappears. The *handwriting* clearly shows this tremor. The lower extremities and the head may show a similar tremor, but not to such a degree as the hands and arms. *Apoplectic attacks* are occasionally observed and these are followed by a transitory paralysis. Such attacks are sometimes the initial manifestations of the disease.

The *sensory symptoms* are not marked, though slight disturbances of touch, pain and temperature-sense may be detected in the fingers and toes or in the distal parts of the limbs; these perversions are usually transitory and changeable. If the disease is associated with hysteria, anesthetic patches or hemianesthesia may exist. Taste, smell and hearing are rarely perverted. The *optic nerve* is frequently affected, the vision being decidedly modified. Optic neuritis is sometimes present and slight atrophy may be discovered. These changes in the visual apparatus are more common in the later stages of the disease and depend, of course, upon the cerebral lesions in the vicinity of the optic nerve or tract. The *pupils* are contracted or unequal, while the reflexes to light and accommodation are occasionally lost. *Nystagmus* or nystagmiform movements are commonly observed.

The *visceral disturbances* are not prominent though sphincter incontinence and gastric crises are occasionally recognized in extreme cases. *Scanning speech* is a marked symptom of cerebral disturbance and is usually detected in the average case. *Vertigo* frequently accompanies this, and if the nystagmus is extreme it may be a troublesome symptom.

The *onset* usually develops slowly, though it may be announced abruptly by an apoplectic attack which is followed by hemiplegia, paraplegia or a monoplegia. The patient usually complains of difficulty in walking or speaking, and the *intentional tremor* is soon noticed. *Pain* is rarely an attending symptom and the disease slowly assumes its chronic character. There are occasional signs of improvement, but these are not usually permanent. Eventually the patient becomes helpless, though death may suddenly intervene as the result of some intercurrent disease.

Diagnosis.—In the classical cases the diagnosis is made by the spastic weakness of the muscles, the cerebello-spasmodic gait, the intention tremor, the nystagmus and the scanning speech. *Paralysis agitans* occurs in older patients, the sensory and special sense symptoms and nystagmus are usually absent, the reflexes are normal, rigidity is permanent, the speech is at first slow and then hurried, the tremor is constant, also involving the head, occasionally checked by voluntary movement, mental phenomena are not usually present, while the immobile face and the propulsive gait are also characteristic. *Tabes dorsalis* is differentiated by the marked incoördination, the loss of knee-jerk, the absence of nystagmus, tremor, ankle-clonus, speech defects, mental disturbances and rigidity, while the fulgurating pains, stiff pupils and the characteristic ataxic gait usually settle the diagnosis. *Hereditary ataxia* begins early in life and the family history of ataxia is usually found. There is no rigidity of muscles, the knee-jerk is lost only as the disease progresses, the motor impairment is purely ataxic and never spasmodic, and there is rarely any optic nerve disturbance. In the *cerebellar type* of family ataxia the reflexes may be exaggerated and optic atrophy may appear, but there is no muscular spasticity. The intention tremor is invariably absent, the speech is slow and irregular but not scanning. *Hysteria* may simulate this disease, but should be differentiated by its stigmata and a careful study of the case.

Prognosis.—The hope of a cure should not be entertained though it is possible to alleviate many of the symptoms by treatment. The tendency to remissions may be regarded as favorable. Westphal has described a pseudosclerosis which closely resembles this disease.

Treatment.—If infection is a factor, we must keep this cause in mind when selecting the remedy. Thus *quinin, iron* and *arsenic* have been highly recommended. For the symptomatic treatment we may favorably consider *zinc phos.*, *zinc picrate*, *strychnia phos.*, and *agaricin.*

PROGRESSIVE MUSCULAR ATROPHY.

This disease is distinguished from the other forms of atrophic development by the progressive atrophy and paralysis of the muscles of the extremities. The cause is found in the trophic impairment and atrophy of the motor cells of the anterior cornu. Its course of development is opposite to that of chronic anterior poliomyelitis.

Etiology.—Adult life furnishes the most cases. It may appear as early as puberty or in rare instances in late adult life. Males are affected most frequently. Syphilis, rheumatism and especially lead poisoning are the most frequent causes, though exposure and functional exhaustion may start the atrophy. Occupations which demand over use of certain groups of muscles, may be etiologic factors.

Pathology.—The process of degeneration is found first in the anterior motor ganglion cells of the cord. A gradual wasting of the whole cornu follows. The anterior roots, the peripheral nerves and, in some cases, parts of the lateral columns are implicated in the degenerative extension. The last feature, however, pertains more particularly to another form of the disease. As the functional control of the muscles is cut off, muscular atrophy and paralysis attend the progress of the disease. Neuroglia and connective tissue development create a sclerosis which may even involve the medulla and brain cortex and the nerve trunks. The posterior cord structure is exempt. The involved muscles undergo fatty metamorphosis with an increase of connective tissue until muscular atrophy and muscular paralysis are coincident with the atrophy of the nerve cells in the anterior cornu.

Symptoms.—Signs of numbness and exhaustion may exist in the affected parts to a slight degree in the beginning. Muscular wasting is the first symptom noticed. This begins generally in the thenar and hypothenar group, and interossei muscles of the hand. The functions of flexion and adduction are imperfect at first and finally lost as atrophy continues. Atrophy of the hand muscles causes the peculiar "ape hand"

and the "griffin claw" appearance. The disease advances upward gradually involving the flexors and extensors of the forearm and shoulder. If one arm is involved first, the other will soon show similar signs of degeneration. The muscular weakness and paralysis go hand in hand with the atrophy. When the "upper arm type" is found the morbid changes attack the deltoid, biceps and triceps and the wasting extends down the arm. The muscles of the back, the hip and the thigh may sometimes be the starting point and then the atrophy and paralysis descend to the legs.

It must be remembered at all times that the paralysis attends or develops with the atrophy. As we observe the progress of the muscular wasting, fibrillary twitchings may be detected and muscular flaccidity or rigidity may be observed. Electrical irritability is lost and contractions may finally develop. Vasomotor disturbances may be present.

Diagnosis.—To make a diagnosis we must differentiate the following diseases: *Chronic anterior poliomyelitis* has generally an acute beginning with the history of a fever; it reaches its height and remains stationary or improves; the *paralysis* also *precedes* the wasting and it affects the muscles in groups according to their physiologic relation. *Lead palsy* has wrist drop, but it involves the extensor muscles only and the interossei seem to escape for a long time; the palsy and atrophy do not show the typical progression as in progressive muscular atrophy. *Neuritis* has more sensory symptoms. *Syringomyelia* has characteristic sensory and trophic symptoms. *Muscular dystrophies* are hereditary, develop slower and have no fibrillary contractions.

Prognosis.—Cure is impossible. There may be some temporary remission in its progress, but it eventually terminates in death as the result of respiratory paralysis following involvement of the medulla.

Treatment.—Measures directed to the general health, particularly when the disease occurs in children and young adults, is very important; an outdoor life should always be encouraged. *Rest* is the next object to be attained, as the wasting muscles should not be overtaxed when the atrophied cell can not give the proper innervation. Massage and exercise are serviceable. *Electricity* is a measure of value; galvanism will doubtless yield the best results; it may be given by applying the positive pole to the cervical region and the negative to the lumbar, and later the weakened muscles may be massaged by the negative sponge. Erb favored its application both to the spinal cord area and the cervical sympathetic. Faradism will give a healthy stimulation in most cases; it is of unusual value to the muscles, which react to the faradic current.

Among the remedies we find that *strychnia* is often of great value in this disease. Gowers claimed for it an unusual efficiency in the early stages. He advised the hypodermatic use, beginning with 1/100 of a grain and gradually increasing to 1/40 or 1/30. It should be given once daily and possibly oftener in extreme cases. The objection to its use is found in the tetanic excitement of the muscles and the undue stimulation which a degenerating nerve will not endure. Internally it may used in the form of *strychnia phos., nux vomica* or *ignatia.*

Plumbum is probably the most frequently indicated remedy. It relates to the pathologic conditions of sclerosis with the subsequent atrophy of muscles, with general emaciation. When the symptoms partake of the characteristics of lead poisoning, it is surely indicated, though the features of colic and other pains may not be present. *Atropin* may relieve the muscular spasms. *Belladonna* may correct any febrile tendency, and overcome the restlessness and nervous excitement which is often present. *Camphor, coffea, hyoscyamus* and *opium* may be studied in this relation. *Arsenicum, ferrum phos., calcarea iodid, china* and *kali phos.* should also be studied.

Argentum nitricum and *aurum mur., gelsemium, arnica, phosphorus, mercurius, physostigma* and *cuprum* may be demanded by their symptomatology.

AMYOTROPHIC LATERAL SCLEROSIS.

The term spastic progressive muscular atrophy has been often applied to this disease. In nearly every respect it is similar to progressive muscular atrophy, except for the additional sclerosis of the pyramidal tract and a corresponding change in some of its symptoms.

Etiology.—It is not so common as progressive muscular atrophy; it afflicts older people and the cause is not so well understood. Syphilis and infectious diseases are not considered as factors; it is probably an idiopathic degeneration of the motor cells of the anterior cord with subsequent involvement of the pyramidal tract fibers.

Pathology.—Atrophic and other changes in the anterior cornu appear the same as in progressive muscular atrophy. In addition to this we find sclerosis of the crossed pyramidal and direct pyramidal tracts. The hypoglossal nucleus and the medulla also show marked sclerotic involvement.

Symptoms.—The *medulla symptoms* appear early; deglutition and speech are difficult. Leg and arm *weakness* follow and the slowly developing *rigidity* is attended by *muscular atrophy* and deformities. The arm, knee and ankle reflexes are exaggerated; the jaw clonus is also present. Sensory and sphincteric symptoms are not marked. The rigidity and contractures are liable to incapacitate the patient in a short time. The *course* of the disease is shorter than in progressive muscular atrophy. Sometimes the bulbar symptoms are most pronounced; then the atrophy of the arms and limbs and the contractures is not so pronounced.

Diagnosis.—In diagnosing this disease we must differentiate transverse myelitis, multiple sclerosis and progressive muscular atrophy. The progressive atrophy plus the rigidity, the exaggerated reflexes and the contractures, together with the complete absence of sensory and sphincter symptoms ought to establish the identity of the disease, particularly if we find bulbar symptoms.

Prognosis.—When bulbar symptoms appear first, the course is short and unfavorable. It is scarcely less unfavorable in the arm and leg type, but life may be prolonged for a greater time.

Treatment.—This is the same as in progressive muscular atrophy.

PROGRESSIVE BULBAR PARALYSIS.

Glosso-labio-laryngeal paralysis or polioencephalitis inferior chronica, is a progressive atrophy and paralysis of the tongue, lips, pharynx and larynx. The condition is caused by the sclerosis of the nuclei of the nerves which supply these parts.

Etiology.—The disease is observed almost always in later life. It may be attributed to the usual causes of sclerotic diseases and may be a feature of amyotrophic lateral sclerosis, tabes, or the wasting diseases. Syphilis, lead poisoning and diphtheria may predispose to it. The overuse of the muscles involved may also predispose by overtaxing their nerve nuclei. The playing of wind instruments and glass blowing have been discussed as causes.

Pathology.—The nuclei of the hypoglossal, the spinal accessory, the facial and the motor part of the trifacial nerves suffer most decidedly from the sclerotic degeneration. The nerve trunks and the muscles which they supply gradually show the effects of a similar degeneration.

Symptoms.—All the symptoms are insidious; the *tongue* is first affected. *Pronunciation* is indistinct and difficult, particularly in the articulation of the lingual consonants. The tongue becomes flabby and gradually loses its muscular power. *Mastication* is difficult because the food can not be rolled in the mouth by the tongue. Finally the tongue can neither be lifted nor protruded. As the orbicularis oris is invaded, the *lips* can not be approximated perfectly; certain vowels and certain consonants can not be properly articulated. The patient can neither blow nor whistle. The lower lip droops, saliva dribbles from the mouth, deglutition is impaired and very soon the mouth can not be closed; the lips are very thin and the lower part of the face becomes motionless and expressionless. The upper part of the face presents a picture of anxiety and suffering.

Atrophy of the palate gives a nasal tone to the voice. The pronunciation of the lingua-palatal consonants is imperfect, and fluids often regurgitate through the nose. *Pharyngeal paralysis* increases the difficulty in deglutition; food enters the respiratory tract and paroxysmal cough ensues. *Pulmonary edema,* cardiac failure and pneumogastric paralysis are now liable to follow. When the *larynx* is paralyzed the abductors are most affected, and as a result the glottis stands wide open and the voice is lost. There may be some remission in the symptoms, but the termination comes in about three years. The mind is but slightly affected, though there is much emotional excitement. The symptoms generally indicate a bilateral involvement of the nuclei.

Diagnosis.—With the symptoms enumerated above, there should be little difficulty in making a diagnosis. There is an asthenic type of bulbar paralysis in which there is no degenerative process found in the nuclei, and there is neither muscular atrophy nor such profound symptoms.

Prognosis.—This is always unfavorable.

Treatment.—This is similar to the other spinal atrophies. Electricity may serve to lessen the extreme symptoms. The laryngeal and pulmonary symptoms will demand careful attention. Otherwise remedies like *atropin, zinc, plumbum, phosphorus* and *argentum nitricum* may be indicated for special conditions.

PSEUDO-MUSCULAR HYPERTROPHY.

This disease is a characteristic progressive muscular dystrophy. While the course of the disease is toward paralysis from atrophy, the typical symptom, in the beginning, is an apparent muscular hypertrophy. It is recognized at first by the evidence of muscular and fatty increase and the progressive weakness of the lower limbs.

Etiology.—There are many reasons for believing it to be a *family disease.* It appears in the early part of life and particularly in young children. During the period of muscular development, fatty infiltration and hypertrophy are more liable to exist; a condition of *malnutrition* naturally contributes to this. If the disease appears later than childhood it is most liable to manifest itself at the time of puberty or subsequent to some of the acute diseases of children. While it does not necessarily follow acute diseases, these may act as exciting causes. The other factors of atrophic diseases do not seem to apply in these cases.

Pathology.—The best authors define the pathologic condition as a degenerative atrophy in distinction to a simple atrophic wasting. In this disease we find various muscular changes. The *muscles* in the parts affected are hypertrophied, the fibers are swollen and the nuclei are increased in number; some fibers may split or break up into fibrillae and in the midst of all these changes there may be signs of atrophy. In time connective tissue increase begins and *progressive muscular* *atrophy* follows. Fatty deposits are found during the early stage of atrophy and, in some cases, lipomas may develop. During the later stages the fat is gradually absorbed and the muscles degenerate into connective tissue masses.

Symptoms.—*Weakness* in the legs and changes in muscular contour are first evident. The patient stumbles easily and has a peculiar swaying or waddling gait. The leg muscles, to all appearances, undergo gradual hypertrophy, and in rare instances there is an increase of adipose tissue over the entire body. These muscles seem to be firm and strong, but they are very weak and lack contractile power.

Following this *pseudo-hypertrophy* we find that certain groups of muscles begin to show signs of *atrophy.* The calf muscles, the extensors of the hip and knee, and the flexors of the hip are among the first to develop the atrophy. As the result of this the shuffling gait is aggravated and the patient is unable to ascend stairs. This weakness of the extensors and flexors of the hips and knees, gives rise to a marked *lordosis* which disappears when sitting. It also explains the typical manner of rising from the dorsal decubitus. The patient cannot get up naturally, but first turns over on the face, then works himself up on his knees, then, with the hands on the floor he straightens his limbs until he assumes the "all fours" position and finally, with one hand at a time, climbs up upon his body until he assumes the erect posture. *Talipes equinus* is liable to follow these contractures of the leg muscles. In extreme cases the legs and arms may be completely flexed.

Sensory disturbances are absent and fibrillary twitchings are rarely observed. The *reflexes* are lost as atrophy develops, but the sphincters are undisturbed. While there are no distinctive trophic changes, retarded growth is always in evidence with the persistent atrophy. In all cases mental apathy is a progressive symptom.

There are *several varieties* of this disease which are named in accordance with the groups of muscles first affected by the pseudo-hypertrophy. For instance, there is a *facio-scapulo-humeral* form which begins in very young children. Here the *atrophy* starts in the face and gives a peculiar appearance known as the "myopathic facies." There is a weakness of the oral muscles which causes the lips to protrude. In addition to this the shoulders and arms are affected in a similar manner. As the atrophy advances the clavicular line is downward, thus giving an apparent lengthening of the neck, and a drooping shoulder. The scapulae protrude and the roundness of the shoulder disappears.

The *juvenile type of Erb* develops very early in childhood. It is confined mostly to the shoulders and upper arm muscles. The forearm and leg are not affected for a long time, if at all; the infra- and supra-spinati invariably escape. The *peroneal type* is another form in which the peronei are first affected and the atrophy gradually extends to the calves and thighs; generally this form is limited to the legs.

Diagnosis.—If the symptoms and the physical characteristics enumerated above are carefully taken into consideration, there will be little confusion with any other disease.

Prognosis.—There is no possibility of a cure in this disease, but we may check its rapid development if we overcome the tendency to malnutrition.

Treatment.—The general care of the patient which embraces moderate exercise, gymnastics and massage, may retard the fatty metamorphosis and, to some extent, correct the atrophic tendency. Electricity does not seem to avail much. We must be on constant guard against the ravages of any infectious disease which may develop. Out-door life and the cold sponge bath will be of great value. The question of prophylaxis is most important. If one child in a family is affected in this way, other children should not be born to this misfortune.

Remedies do not accomplish much when the disease is well established. In the beginning, however, we ought to gain something by their use. *Phosphorus* gives the clearest picture of the fatty degeneration, and should be employed for a long time in the incipient case. *Calcarea carb., calcarea phos.* and *kali phos.* may also be demanded in this disease. *Arsenic* will be indicated when anemia is typified by exhaustion. *China* is useful when septic symptoms are added to the general weakness.

TUMOR OF THE SPINAL CORD AND MENINGES.

Tumors which involve the spinal cord primarily are rare. The majority start in the meninges. A few may originate in the vertebrae. The general symptomatology is similar in most cases. It depends much upon the location, character and size of the tumor. When the cord alone is affected, the growth is generally small and single; multiple sarcoma is an exception to this. As the meninges are more frequently involved the study of growths here may be properly grouped under the same head.

Etiology.—Most tumors appear during middle life; tubercle alone may develop early, though lipoma is claimed to be a congenital possibility. Males furnish a great majority of the cases. Traumatism and syphilis are the predisposing causes.

Pathology.—Glioma and sarcoma are the most common forms; true carcinoma is rare. Fibroma, myxoma, gumma and tubercles are liable to occur. Most of these start in the extradural tissue or in the meninges; they invade the cord structure by compression, creating pressure atrophy or, if inflammation is present, softening and a general myelitis. The glioma, by its wide diffusion, may form cavities. Sarcoma may infiltrate the cord and meninges to a considerable extent. The characteristic histological features which typify these growths in other parts of the body are recognized when the cord is involved. The lower cervical, upper and lower dorsal areas are the usual locations.

Symptoms.—*Pain* is constant and severe; it appears early, and at first causes a *girdle sensation* by irritation of the posterior roots, and follows the distribution of the peripheral nerves. As the *sensory tracts* are invaded it is referred to parts below the lesion. *Numbness,* hyperesthesia and later anesthesia may be experienced. All sensory symptoms are detected on one side more than the other, though they may be bilateral.

When the *dorsal* or lower areas of the cord are affected *paraplegia,* with exaggeration of reflexes, is present; hemiparaplegia may precede this. Should a *lumbar* segment be the seat of invasion, the *reflexes* are lost and the sphincters are paralyzed early. The location of a tumor is settled by a knowledge of cord localization.

Brown-Séquard's paralysis illustrates the average symptomatology of a cord tumor. This is represented by a paralysis of motion and muscle sense on the side of the tumor, and loss of pain and temperature sense on the opposite side. It is due to a tumor limited to one side of the cord and involving the posterior horn completely, the anterior horn slightly, and the lateral white structure to a considerable extent. Thus hemiparaplegia and cutaneous disturbances on the opposite side are its characteristic symptoms.

The symptoms are different when the tumor is inside or outside of the dura. When *within the dura* hemiparaplegia is common, but pain, spasm and rigidity are less; when *without the dura* hemiparaplegia is less frequent, but pain and motor disturbances are more pronounced.

Diagnosis.—We must distinguish between cord tumor, vertebral caries, transverse myelitis and hypertrophic pachymeningitis. *Syphiloma* will be diagnosed only by the history and the fact that it responds to antisyphilitic treatment. In the average case it is safe to decide on sarcoma or glioma.

Prognosis.—This is always unfavorable, though surgical measures may help some cases. Syphiloma may be relieved by remedies.

Treatment.—In *extradural tumors* surgical relief is possible. Symptomatic treatment is the only alternative we have. When syphilis is the cause we should prescribe such remedies as *potassium iodid* and *mercury.* When tuberculosis is present, we should direct our attention to that disease. It is possible that *arsenicum iodid, aurum mur.* and *argentum nitricum* may be of some use.

CAVITIES IN THE SPINAL CORD.

Under this head we are to consider various deformities of the spinal cord in which the tissue degeneration has permitted the formation of cavities. The morbid process may occur independently, or in association with other diseases. The first of these is *hydromyelia.* This is a dilatation of the central canal with the cavity filled with fluid. It is most frequently associated with hydrocephalus or spina bifida. It is not generally extensive, though in rare cases it may pervade a considerable portion of the cord. *Myelitic cavities* are the result of minute hemorrhages and the subsequent myelitic softening. They are usually small and are distributed in different parts of the cord. The most important of these is *syringomyelia,* and as it deserves more extensive study it is given a separate heading.

SYRINGOMYELIA.

This is a disease of the spinal cord characterized by embryonic defects of the central canal and the formation of moderate or large sized cavities. These cavities are invaded by gliomatous tissue and this pathologic condition gives rise to peculiar sensory and trophic disturbances.

Etiology.—The disease is a rare one and develops in early life. The predisposing causes are not well understood and the etiology is not clearly established.

Pathology.—A cross section of the cord reveals an irregularly formed cavity extending upward and downward. These cavities are of various shapes and sizes; in some cases they may extend to the cord periphery. The cervical area is the most frequent location and they invade the posterior horns more than the anterior, though, in large excavations the anterior structures may also be invaded. The cavity is filled with cerebrospinal fluid and gliomatous tissue. The hyperplasia of neuroglia cells is evidently the primary pathologic development. This takes place in connection with the embryologic defect in the formation of the central canal. The imperfectly contracted canal, supplemented by the gliomatous development, permits the formation of a larger hole in the cord during the development of the child. A tumor may be found when the gliomatous increase is excessive.

Symptoms.—The symptoms are not always well defined. Many cases are diagnosed only at autopsy. The typical symptoms are as follows: The *onset* is insidious. *Pain* in the back of the neck and arms is accompanied by paresthesia, numbness and atrophy in the hand muscles; the wasting gradually extends to the trunk. *Fibrillary contractions* and partial electric degenerations are in evidence. *Anesthesia* to temperature and pain is detected in the hand and arm; the sensation of touch is not disturbed. *Paralysis* and deformities in the upper extremity naturally follow. As the muscles of the back suffer from atrophy, a *scoliosis* appears in the dorso-lumbar region. A *spastic paraplegia* develops gradually as the leg muscles are affected. Most of the paralytic symptoms, however, appear in the upper part of the body.

The *trophic perversions* are pronounced. The hands are red and edematous; the skin is either abnormally dry or moist;

eruptions in the form of bullae, eczema or herpes are present, and ulcers or gangrene are not uncommon. The same trophic change may pervade the osseous structure, giving rise to arthropathies and spontaneous fracture. The disease pursues a slow course and the pathologic condition may involve the medulla. The sphincters may be slightly disturbed.

Diagnosis.—The diagnosis should be settled by the cardinal symptoms—a progressive muscular atrophy, the anesthesia to temperature and pain, but not to touch, the scoliosis and the characteristic trophic disturbances.

Prognosis.—There is no possible help in a typical case. The remission of symptoms may give some temporary encouragement.

Treatment.—As this disease starts with a congenital defect, there seems to be no possible help from treatment, unless we may find remedies which will overcome the increase of gliomatous tissue. *Arsenic, argentum nitricum* or *chlorid of gold* may possibly aid this effort, if given long enough. The trophic function may be helped by such remedies as *silicea, natrum sulph., graphites* and *thuja.*

Various Types.—The symptoms of this disease vary in accordance with the location and extent of the cavity in the spinal cord. There may be symptoms which simulate *chronic myelitis* when the destruction of tissue is diffuse. The *bulbar involvement* may be extreme, giving rise to symptoms from cranial nerve implication. *Morvan's disease* represents a peculiar form of syringomyelia complicated by a peripheral neuritis. It is characterized by painless whitlows on the ends of the fingers of both hands. The trophic debility is so extreme that ulceration and gangrenous degeneration may take place. The nails are very brittle and may drop off. The joints may be somewhat affected.

DISEASES OF THE BRAIN AND ITS MEMBRANES.

Pachymeningitis Externa.—Inflammation of the outer layer of the dura is the more common form. It may be secondary to traumatism or disease of the cranial bones. It may follow fractures or surgical operations upon the head. Caries, necrosis, or syphilis invading the softer osseous structure of the cranium may be primary causes.

Symptoms.—Severe headache, fever and delirium appear early after an injury. The head is very sensitive to touch and stupor, convulsions or paralysis may soon follow. Suppuration and the signs of sepsis rapidly develop. It runs an acute or subacute course.

Prognosis.—This depends upon the extent of the lesion and the surgical relief.

Treatment.—Surgical measures offer about the only treatment. Cases due to syphilis may demand both surgical attention and antisyphilitic remedies.

Pachymeningitis Interna.—This variety is of rare occurrence. It is an inflammation secondary to dural hemorrhage, or may follow a protracted infectious fever. It is claimed by some authors that it may be associated with the general paralysis of the insane and chronic alcoholism. Degeneration of the blood clots takes place between the dura and the arachnoid.

Symptoms.—The only characteristic symptoms are defined by slight apoplectiform seizures. General exhaustion, optic neuritis, nystagmus and mental derangements are possible symptoms. When it occurs in childhood, convulsions or hemiplegia may mark its presence.

Prognosis.—The prognosis is unfavorable, and about the only treatment which will avail anything must be directed toward the control of hemorrhage.

Acute Leptomeningitis.—This term refers to an acute simple inflammation of the pia mater in contradistinction to epidemic cerebrospinal and tuberculous meningitis. Brain fever is a common definition.

Etiology.—Traumatism is a frequent cause. Diseases of the middle ear, frontal sinuses and nasal passages are predisposing factors. It most commonly occurs secondary to infection. Pneumonia, variola, typhoid fever, rheumatic fever, whooping cough, measles and scarlet fever most frequently contribute the infection. Endocarditis, empyema, brain neuroses, gout and renal disease are rare causes. It may exist at any age, but it is oftener seen among children.

Pathology.—The pia is thickened by the purulent or fibropurulent exudate; this is noticed mostly at the base of the brain, though it may be limited to the convexity. The ventricles and the arachnoid cavities show an increased amount of fluid which may be turbid. The blood vessels are injected and the inflammatory process may affect the cerebral surfaces. The micro-organisms are those of the disease which contributes the infection.

Symptoms.—*Prodromal* symptoms are often unobserved. The disease is ushered in by the sudden appearance of *rigors,* severe headache, fever and delirium. The headache persists with exacerbations. *Vomiting* is of violent or explosive character, though it is not always present. The neck is rigid and the head is drawn back. Often this *rigidity* affects the whole body and epileptiform convulsions magnify this. The *abdomen* is retracted and the evidence of a very grave illness is at hand.

Vertigo is often present, the skin is hyperesthetic, the face is flushed and the patient is sensitive to the slightest noise. A dull instrument drawn quickly across the skin leaves a red line (tache cérébrale). The *pupils* are contracted and uneven. The *fever* is irregular and varies from 101°-104° F. *Respiration* is rapid and irregular.

As the acute features subside *stupor* or coma supplant the delirium. *Rigidity* may remain, though somewhat modified. The *abdomen* is still retracted, but the *pupils* are dilated and the skin becomes moist with the disappearance of the fever. The *sphincters* may act involuntarily and a paralytic stage may intervene in which the patient is liable to die.

When the pia is affected more at the *base* of the brain the delirium is modified, but *optic neuritis,* cranial nerve palsies, vomiting and retraction of the head are more pronounced. The disease generally runs a course of one to two weeks, though a patient may die from coma in the early stage.

Diagnosis.—The sudden onset, the vomiting, retraction of the head and rigidity, the contracted pupils which later are dilated, the retraction of the abdomen and the delirium leave no doubt as to the diagnosis.

Prognosis.—The disease is always a grave one. It depends upon the infection or injury and the course it takes. If the symptoms show any tendency to gradually subside under careful treatment, some hope may be entertained.

Treatment.—To a certain extent, surgery may be demanded. Injury of the skull and mastoid disease call for these measures. Diseases of the ear and nasal sinuses must also be looked after. We should seek, in every predisposing disease, to prevent the meningeal infection. The patient should be kept absolutely quiet. The *icebag,* applied to the head, will give the greatest relief. The *diet* should be light and nutritious. Delirium may make rectal feeding necessary. *Catharsis* is often required and may be induced by small doses of salines, cascara or calomel in divided doses. The use of the *normal salt enema* should not be overlooked. *Good nursing* is of highest importance in this disease. *Opiates* and hypnotics may be necessary. *Aconite, arnica* and *hamamelis* are often indicated in the beginning. *Belladonna, hyoscyamus* and *stramonium* will cover the usual congestive features, particularly when delirium is present. *Bryonia, gelsemium, glonoin, hellebore* and *anacardium* will often meet the symptoms presented here.

TUBERCULOUS MENINGITIS.

The bacillus tuberculosis is the cause of this form of leptomeningitis. The infection causes inflammation which is most intense at the base of the brain, the exudate is not purulent and is invariably attended by distention of the ventricles, which gives rise to the term "acute hydrocephalus."

Etiology.—Tuberculous meningitis is almost always found in children between the ages of two and twelve. Heredity, environment, occupation and all the predisposing factors which relate to other tuberculous conditions are found in these cases. Tuberculous infection is frequently observed in other organs. Traumatism has recently been referred to as a cause, inasmuch as trifling injuries of the spine and head have seemed to favor the rapid development of the disease. The eruptive fevers have also been considered as predisposing causes.

Pathology.—At the base of the brain we find a jelly-like exudate, and tubercles in various stages of degeneration. These, together with a fibrinous deposit, are limited to the area of the blood vessels and vascular spaces. This exudate reaches the convexity of the cerebrum through the fissure of Sylvius and the smaller sulci, but the greatest deposit is at the base. While the inflammatory invasion may affect a considerable portion of the brain tissue, the tubercles remain in patches about the arterioles and larger vessels. The choroid plexus and ventricles are finally invaded, giving rise to their dilatation and the increase of ventricular fluid; this, with the additional increase of arachnoid fluid, gives the symptoms of acute hydrocephalus. The bacillus tuberculosis is always present in these nodules; sometimes a large tuberculous mass may exist. Mixed infection is uncommon, hence the absence of pus. Not infrequently tuberculosis elsewhere in the body is discovered.

Symptoms.—The *early symptoms* may not be significant of the real disease. A prodromal malaise is attended by distressing headache, digestive disorders and some fever; the child may be at first fretful, apathetic and later drowsy or affected by marked stupor. This condition may remain for some time, during which physical exhaustion and loss of flesh may be significant of a serious disease. When the *cerebral symptoms* appear, paroxysmal and severe headache, explosive vomiting and constipation are manifest; the sleep is often disturbed by the hydrocephalic cry; there is extreme rigidity of the neck, with a tendency to slight opisthotonos, the thighs are flexed, and Kernig's sign and abdominal retraction are manifested. The *temperature,* though rarely high, may suddenly rise, Cheyne-Stokes' respiration may appear preceding the paralytic and comatose state. *Emaciation* progresses rapidly. Remission of the typical symptoms may take place, a period of apparent improvement being usually succeeded by the intensification of all the classical symptoms. Just prior to the fatal termination the fever may be very high (106°-108° F.), the pulse and respiration also being greatly accelerated.

Vasomotor disturbances are among the early features of the disease. Alternate flushing of the face and pallor are attended by the typical *tache cérébrale.* On account of the basilar involvement, limitations of the sight may follow fundus changes. *Cranial nerve* implication is represented by the frequent appearance of strabismus. As the third nerve is affected the pupils are at first contracted and rigid, while later they are dilated. *Facial paresis* follows facial nerve compression, and the tongue, pharynx and larynx are affected from a similar cause. The *mental state* represents the various features of apathy, delirium or coma; the patient always resists disturbance. All the symptoms may vary in intensity and character, dependent, of course, upon the localization and extent of the exudate. The general health and heredity have much to do with this. The disease may run a subacute course, or a fatal termination may occur from the exaggerated conditions of the first period. Ordinarily the extreme symptoms subside after a few weeks, only to be followed by a sudden and marked remission, during which death takes place.

Diagnosis.—A positive diagnosis in the early stages is impossible, unless the symptoms are clearly defined. The characteristic vomiting, with severe headache and the other signs of brain irritation should be recognized. The hydrocephalic cry, the stupor and pressure symptoms, together with the tendency to remission of symptoms, should aid us in making the diagnosis. The discovery of tuberculous invasion elsewhere, the heredity and the emaciation should be suggestive signs. The subacute character of the symptoms will help to differentiate *acute meningitis.* If tubercles are found in the retina, or if they can be obtained by spinal puncture, the diagnosis will be confirmed.

Prognosis.—While the disease may manifest variations of symptoms, it usually terminates in death, though cases of cure are reported by competent observers.

Treatment.—The same *general care* as applied to acute meningitis should be resorted to here. *Sedatives* will often be demanded and for this purpose chloral or bromid of potassium are probably the best. The icebag or the ice-coil are indicated for the inflammation and pain and to lessen effusion. Hot applications or the hot bath may be more agreeable to some patients. The high normal salt enema and the systematic cool sponge also have their place. Quincke's lumbar puncture has been resorted to for relief of intracranial pressure, though this gives usually only temporary relief. The *diet* should be highly nutritious, eggs, milk and meat juices being especially valuable.

From the *remedial standpoint* we should first of all consider the tuberculous cause. The treatment which generally applies to tuberculosis elsewhere may be followed here. The iodin and creosote preparations should always be considered; guaiacol is probably the best. The internal use of iodoform, in the second or third decimal potency, has been recently suggested, but its use is more serviceable in mixed infections. For the acute symptoms *gelsemium, baptisia, belladonna* and *cimicifuga* should be employed. For the effusion *hellebore, cantharis, bryonia* and *apis* may be indicated. The use of *tuberculinum* is suggested, though the results with this remedy, in my experience with tuberculous affections, have not been encouraging. Iodoform inunctions are highly recommended, a number of cures being attributed to their employment.

CHRONIC HYDROCEPHALUS.

This disease refers to the great increase of cerebrospinal fluid within the ventricles, the gradual enlargement of the head and pressure symptoms. It is either congenital or appears during infancy. Mechanical causes or structural defects are the primary disturbances; nutritional perversion may be a factor. Subdural or external hydrocephalus has also been recognized.

Etiology.—The usual period of development is at birth. Syphilis and alcoholism on the part of the parents are predisposing factors. Rickets and other dyscrasiae are contributing causes. Traumatism during birth or after may incite it.

Pathology.—A colorless fluid of low specific gravity accumulates to a considerable extent in the ventricles of the brain. This may exceed two gallons in amount. The aqueduct of Sylvius and the foramen of Magendie may be occluded by inflammatory obstruction, thus preventing the escape of this fluid into the general arachnoid cavity. The distention of the ventricles pushes out the cerebral walls so that the transverse diameter of the head is greatly increased. The ventricular lining is hypertrophied, but the cranial bones are thin as the result of pressure from within and malnutrition.

Symptoms.—If the disease is congenital the *head* may be so large at birth that instrumental delivery or craniotomy is necessary. It more frequently develops gradually during the first few months after birth. The head rapidly or slowly enlarges in all directions. The smallness of the face presents a strange contrast to the bulging head. The cranial sutures do not approximate, the venous circulation is interrupted, optic atrophy develops, nystagmus and strabismus may be present and fluctuation may sometimes be detected. The *child* becomes peevish and restless, general nutrition is perverted and the growth is retarded. The *mental powers* do not develop, the child can not walk and the optic atrophy tends toward blindness. The disease may pass slowly into a chronic condition, which may last for years with varying conditions. Often the child lives on in this chronic state, slowly gaining some of its mental and physical powers. As a rule, however, the body is dwarfed and the mind is that of an imbecile. The pressure may eventually cause vomiting, coma and convulsions, or the child may die from other causes.

Diagnosis.—The characteristic symptoms will easily settle the diagnosis. *Rickets* simulates it, but the head is square, hydrocephalus is not noticed, the enlargement is less, and involvement of other bones is observed.

Prognosis.—In some cases a fairly hopeful result may be expected. If the disease develops in early childhood, the physical development may gradually overcome it. Congenital cases are usually fatal.

Treatment.—The best results are obtained by careful regard to diet, hygiene and by the use of such remedies as *calcarea carb., calcarea phos., silicea, apis, hellebore, iodoform, arsenicum iodid* and *sulphur.* Surgical measures have not yielded good results.

CEREBRAL HYPEREMIA.

An undue amount of blood may invade the brain from physiologic or pathologic causes. Sunstroke, drug irritants, alcohol and injury contribute to the latter condition. Mechanical causes which prevent the return flow may also incite it. Fever, mania and meningitis favor it. Organic diseases of the heart and other organs may also induce it. The *symptoms* may be defined by the sense of fulness or pressure, headache, vertigo, mental excitement, insomnia, etc. The disease generally yields to local and remedial measures.

CEREBRAL ANEMIA.

This condition is also acute or chronic as the result of various diseases and conditions. General anemia is the most frequent cause. It may be due to organic changes in the cerebral arteries, to syphilis, Bright's disease, profuse hemorrhages and disorders of nutrition. Fright is a rare cause.

The *symptoms* are vertigo, nausea, pallor, confusion of ideas and syncope. In the *chronic form* mental apathy, somnolence, mental depression and headache are present. The *diagnosis* is based on the symptoms just mentioned.

The treatment embraces the improvement of the general nutrition by diet, hygiene and the use of such remedies as *iron, arsenic, strychnia* and other tonics.

ABSCESS OF THE BRAIN.

Brain abscess is a localized suppurative inflammation of the cerebral structure. The cause is found in the introduction of pyogenic bacteria.

Etiology.—Any infection which produces cerebral inflammation may be considered a cause. Suppurative ear disease, caries of the ethmoid and other cranial bones are the most frequent factors. Injuries or fractures of the cranial bones may, by the contusion of tissue and subsequent infection, establish a localized suppuration. Diseases of the lungs and general pyemia furnish the suppurative cause in many cases. Pulmonary tuberculosis, purulent bronchitis and empyema may be especially mentioned. Septic endocarditis is a rare cause. The infectious fevers, such as influenza, typhoid, diphtheria, erysipelas and variola are etiologic factors. The mixed infection which is liable to attend any of the cerebral tumors may be the only cause.

Pathology.—The abscess is generally single and may attain considerable size, containing several ounces of pus. Usually the large ones are located in the occipital or frontal lobes. The most common seat is in the temporo-sphenoidal area and here they are of moderate size. Sometimes the abscesses are multiple; this is particularly true in the general pyemic state. The congestive sign is recognized early by the intense redness of the cerebral tissue affected. This was once termed "red softening," but it is only a primary feature of the suppurative process. As the inflammation continues, destruction of nerve fibres and cells take place; neuroglia tissue in the part involved transplants the normal structure; granular corpuscles and an excess of leukocytes soon make up the purulent mass which is walled off by a fibrous structure until it becomes completely encysted. There may be a communication through a fistulous tract with the diseased bone. The gray matter of the cortex is not so frequently affected as the white substance. The pus varies in accordance with the extent of the lesion; in the later stages it becomes thick and fetid as in any abscess. The streptococcus and other pyogenic bacteria are generally in evidence.

The cerebellum, pons and medulla may rarely be the seat of the abscess. The region of the middle cerebral artery is the point most often involved when the infection is derived through the circulation. Idiopathic brain abscesses are said to be located always in the frontal lobe.

Symptoms.—The character of the symptoms depends upon the location, rapidity of the abscess formation and the virulence of the infection. The disease may run an acute or chronic course. In many cases the clinical signs may be so obscure that a diagnosis can not be made. In the *early stage* the temperature may be slightly elevated, but it is more frequently subnormal. *Rigors,* profuse sweats, the irregular fever and great prostration manifest the toxic condition. *Headache* is constant and severe; vomiting, attended by constipation or diarrhea, mental torpor which may end in delirium or coma, and the occasional presence of convulsions are typical symptoms. These features may last a few days or weeks, when there is liable to be a stage of remission. The patient now gives signs of recovery, though there may be a reappearance of the cardinal symptoms described above. As the abscess development progresses, *rupture* of the limiting capsule and general infiltration of the cerebral structure are liable to take place. This condition is ushered in by the sudden appearance of an apoplectic stroke and the subsequent presence of hemiplegia. Usually death comes at this time, though the hemiplegia, with its characteristic features, may be a progressive development. It is possible for the chronic condition to continue for some time, with occasional symptomatic exacerbations. The inevitable return and sudden exaggeration of the typical symptoms will, however, eventually end in death.

The *focal symptoms* are often well defined and enable us to locate the abscess. If embolism of the cerebral arteries exists, hemiplegia may be an early and positive sign. The circumscribed headache does not always define the accurate location of the pus. We may depend much upon the contributing factor in finding the lesion, but as the majority of abscesses develop in the frontal, temporal and occipital lobes, the cranial nerves are slightly affected and manifest few symptoms of importance. *Multiple abscesses* will be even more confusing. Hemianopsia, Jacksonian epilepsy, optic neuritis, aphasia and other signs of a true cerebral localization may, in certain cases, give some definite focal indications. *Local tenderness* of the scalp has been regarded by some observers as a guiding sign.

Diagnosis.—The contributing cause, the symptoms of sepsis, the headache, focal symptoms, vomiting, irregular or subnormal temperature, the hebetude and delirium or coma will be differentiating points for consideration. *Tumors of the brain,* meningitis and phlebitis of the sinuses must be excluded.

Prognosis.—There is no possibility of a cure except by surgical measures. This is not assuring, from the fact that secondary sepsis may follow, even though the abscess may be located and safely reached.

Treatment.—The use of internal remedies avails us nothing so long as the pus is encapsulated and continues to form. Some encouragement has been given by surgical procedure.

ACUTE EXUDATIVE ENCEPHALITIS.

This is an inflammation characterized by an exudate which affects the gray matter of "definite cerebral structures." In one form known as *acute polio-encephalitis superior* the exudate invades the nerve nuclei of the eye muscles, giving rise to muscular disturbances defined as ophthalmoplegia. In the *acute polio-encephalitis inferior* the exudate involves cranial centers in a lower part of the medulla, giving rise to glosso-labio-laryngeal palsy or bulbar paralysis.

Another form of this disease has been described by Strümpell in which the exudate is found in the cortex and causes cerebral palsies of children. This form is also termed acute cortical polio-encephalitis. None of these diseases is sufficiently prominent to demand any extended consideration.

ACUTE EXUDATIVE ENCEPHALITIS WITH HEMORRHAGE.

It has been found that a form of encephalitis exists in which the inflammatory invasion of the brain is secondary to minute non-suppurative hemorrhagic foci. It is often mistaken for meningitis.

Etiology.—Infectious diseases, particularly influenza, are the most frequent exciting causes. It may depend upon alcoholism, septic endocarditis and possibly the puerperal state. While it may occur after the twentieth year, it is more often observed during childhood.

Pathology.—Numerous but minute hemorrhages are found in the cerebral white matter and the basal ganglia. Intense congestion and inflammation and some tissue softening exist in the vicinity of the hemorrhagic foci. The cerebral fluid is increased and blood stained, the ventricles are slightly distended and the surrounding blood vessels are dilated. Connective and neuroglia tissue increase may develop a sclerotic condition. Larger hemorrhages may lead to apoplexy. The influenza bacillus has been found in the infiltrated brain tissue.

Symptoms.—The symptoms in the beginning simulate those of meningitis. Following an acute infection, headache, high fever, vomiting, convulsions or localized palsies suddenly appear. Photophobia, delirium, hebetude or stupor are soon recognized. The pulse is rapid and respirations are frequent and shallow. The stupor now gradually or rapidly deepens, or there may be a semiconscious condition for days. A restless and irritable state may succeed gradual improvement in the stupor until a period of convalescence is reached. Recovery is possible, though features of epilepsy, aphasia or paralysis may intervene in accordance with the extent and local disturbance in the brain.

Diagnosis.—We must compare this disease with *meningitis.* It does not show the projectile vomiting, the hyperesthesia, the small pupils, nor the rigidity of the neck and limbs. The onset is also sudden and the coma or semi-coma is characteristic.

Treatment.—The care and consideration which are given in the early conditions of cerebral hemorrhage or meningitis must be observed. The patient must be put to bed and kept very quiet. The bowels and kidneys should be helped to act freely by the use of saline cathartics. The fever should be treated by remedies and systematic sponging, as in meningitis or other febrile conditions.

CEREBRAL HEMORRHAGE (HEMIPLEGIA).

Cerebral hemorrhage or apoplexy is characterized by prodromal symptoms, sudden loss of consciousness and by the development of hemiplegia.

Etiology.—The conditions which favor rupture of the cerebral arteries may be found in a high blood pressure with increased weakness of the arterial wall. Arteriosclerosis with increased cardiac action such as accompanies cardiac hypertrophy is the chief predisposing cause. Other predisposing factors are *advanced age,* renal disease, gout, chronic alcoholism, syphilis and possibly rheumatism. All *acute infections* which favor the development of arteritis must be looked upon as causes. Blood diseases may contribute to its development. *Heredity* has its influence and the "apoplectic habit" seems to be established in some families. It is essentially a disease of adult or later life, though traumatism at birth or in early life may cause intracranial hemorrhage. The *inciting causes* may be discovered in sudden physical exertion, shock, violent sneezing, coughing, vomiting, straining at stool and excessive coitus. Those who overwork, eat excessively and indulge in dissipation invite this disease.

Pathology.—The hemorrhage is most frequently central, involving the vessels which supply the basal ganglia, internal capsule and white matter of the cerebrum. While it may occur in the meninges, the pons, or the medulla, the cerebrum is affected in the great majority of cases. The pathology consists in the presence of blood in the brain substance. It may be extensive, penetrating the ventricle, or it may be only enough to separate the nerve fibers. After a time the blood grows lighter in color, the walls of the hematoma showing a reactive inflammation. The pathology of the predisposing disease is present. When the hemorrhage is extensive enough to cause marked and protracted paralysis, degeneration of the motor tract takes place.

Symptoms.—The *cardinal features* of cerebral hemorrhage appear so abruptly that the term "apoplectic stroke" characterizes the onset. The *prodromal symptoms,* though not of frequent occurrence, may consist of numbness of the hand and foot on one side, slight dizziness, headache or fulness in the head, with cardiac irregularity.

The *attack* usually begins with sudden loss of consciousness though convulsions may, in some cases, precede. With the loss of consciousness the patient falls as if in a fit; the *face* is highly congested, the breathing is stertorous, the *pupils* are sluggish, usually contracted or uneven, the *mouth* is drawn to one side, while the cheek flaps with each respiration. The *eyes* are partially closed and the eyeballs are fixed or deviated to the paralyzed side. *Sensibility* is abolished and the limbs are limp, while hemiplegia gradually develops. The *sphincters* are relaxed and involuntary urination or defecation may occur. The *pulse* is hard and slow and the temperature may be subnormal at first, later rising above normal.

If *death* does not occur during the seizure, the coma gradually deepens or consciousness partially returns. A persistent coma, with the Cheyne-Stokes' respiration, delirium and a high temperature presage a fatal termination. Hypostatic pneumonia may complicate the recovery.

In the *majority of cases* the severe signs of shock pass away in a few hours or a few days. At this time hemiplegia is completely established, the palsy being flaccid, with lessened reflexes. The *facial nerve* is not always affected. *Deglutition* and *speech* are imperfect, the *tongue* is paralyzed or, if it can be protruded, it deviates to the affected side. A motor or sensory *aphasia* may be observed. As the motor power of the tongue returns, the speech is thickened and inarticulate. *Facial asymmetry* exists as the result of unilateral paralysis. The conjugate deviation of the head and eyes disappears in a short time. Hemianesthesia, hemianopsia and other sensory perversions disappear in a few days.

The *hemiplegic state* is the natural sequence of the cerebral hemorrhage. The improvement of the symptoms enumerated above continues for a few weeks, until the residual hemiplegia is observed. Slight voluntary motion may now be recognized in the lower extremity and face. The tongue, though deviating somewhat to the paralyzed side, may be protruded, and slight return of the speech is manifest. Perfect articulation is impossible and the patient pronounces certain consonants with difficulty. The *mental faculties* return slowly, though hebetude or melancholia may supervene. The flaccidity of the affected muscles now gives place to increasing rigidity. The reflexes are exaggerated and clonus is in evidence. If careful treatment is not instituted, contractions are liable to follow. The extensors of the foot are usually paralyzed; the heel is drawn up and, when the patient attempts to walk the foot is dragged, the toe scraping the floor. In the *upper extremity* the flexors draw the fingers tightly into the palm of the hand, the wrist is firmly flexed and the elbow is fixed at an angle, while the arm is held closely to the side. The range of joint movement is greatly limited. The contraction of the unaffected facial muscles gives rise to marked asymmetry. Trophic disturbances are not pronounced, unless there is complete inactivity of the affected muscles for a long time. *Vasomotor perversions* may be experienced in certain cases. The mental conditions are often most perplexing. The patient may, at times, be decidedly irritable or periods of acute melancholia may exist. The memory is faulty and the mental deficiency may lead to insanity. Post-hemiplegic epilepsy may occur, particularly in the younger patients.

As the hemiplegia persists and as irregular involuntary movements return, a peculiar set of symptoms may arise. These consist of associated movements, tremor, ataxia, choreic, continuous or athetoid movements. The muscles are generally spastic in their action and cramps are liable to be disturbing features. The electric reactions are not pronounced. The course which this disease pursues varies in accordance with the size and location of the hemorrhage, as well as with the age and physical condition of the patient. The characteristic symptoms as defined above usually prevail.

Diagnosis.—The sudden loss of consciousness, the subsequent hemiplegia and the attending symptoms enumerated ought to be sufficient for a diagnosis. *Meningeal hemorrhage* will generally confuse us at first. This has many similar features, but it usually follows injury, the paralysis develops slowly and there are distinct intermissions in the characteristic symptoms. These differentiated points may be further emphasized by a careful study of this disease. *Cerebellar apo-*

plexy does not generally display so much mental disturbance or such pronounced loss of consciousness. The horizontal position creates vertigo, vomiting and extreme incoördination, but distinct hemiplegia is not always observed. Serious respiratory disturbances are apt to be followed by sudden death when hemorrhage involves the fourth ventricle. *Alcoholic intoxication* may simulate cerebral hemorrhage, or it may be the cause of it. The odor of the breath, the more natural sleep and the fact that the individual can be aroused, are differentiating signs. *Opium poisoning* shows equally contracted pupils, slower respiration and the absence of hemiplegia. *Uremic coma* is more gradual in its development, paralysis is not a feature, and the presence of albumin and casts, together with other signs of renal involvement, will aid in the differentiation. *The epileptic attack* will be excluded by the aura, the sudden cry, the fall, the loss of consciousness, the biting of the tongue, the absence of hemiplegia and the peculiar tonic and clonic spasms. *Hysteria* may be excluded by its stigmata, localized anesthesias, and the characteristic idiosyncrasies which attend its attacks.

Prognosis.—The average case recovers from the first attack but a recurrence is always unfavorable. The patient may live for a long time suffering only as a hemiplegic. Improvement is at best very slow. A stationary condition in the paralysis is generally reached after a few months. The limb may be used, but it keeps its hemiplegic swing and more or less rigidity. The arm paralysis is rarely cured. In the early period much depends upon the coma. A profound and persistent coma or delirium is always unfavorable. High fever or persistent subnormal temperature are also grave signs. Alcoholism, nephritis, extreme arteriosclerosis and syphilis accentuate the danger. Pons and cerebellar hemorrhages are decidedly unfavorable.

Treatment.—This must first consider the attack alone. Here we have an opportunity to minimize the after results by judicious attention to the hemorrhage. The patient must be kept absolutely quiet in bed with the head elevated and free from excitement. The greatest danger is the anxiety of friends who seek to do something to overcome the shock and paralysis. An *icebag* applied to the head and a hot water bag to the feet are the best primary measures. *Free catharsis* should be induced; croton oil (m. 1-2), or elaterin (gr. 1/16-1/10), and an enema should be given. *Venesection* in plethoric individuals may be demanded to reduce the arterial tension. Our principal aim should be to prevent the recurrence of hemorrhage. For this purpose, in addition to the icebag and the enforced rest and quietude, our treatment will be symptomatic. *Hamamelis, arnica, ipecac, secale* and *china* will be of service in this respect. The nervous state and the excited condition of the circulation in the early stage will demand the use of *aconite,* which may be the only remedy needed for some time. *Belladonna* represents the characteristic cerebral congestion, which is evidenced by the full bounding arteries, the flushed face, and the general vasomotor perversion. *Opium* may be required when there are signs of recurrent bleeding or marked venous congestion. *Glonoin,* in the third decimal potency, will have great influence in overcoming the high arterial tension. *Veratrum viride* will do this to a certain extent, though there is greater danger of cardiac depression from its use. *Strychnia* and other heart stimulants like *crategus, digitalis, adonis, convallaria* or *strophanthus* may be necessary adjuvants to maintain the cardiac regularity. The main remedial effort in the beginning should be to check the hemorrhage, quiet the nervous excitement and then to encourage the rapid absorption of the exudate. The *nervous symptoms* following the first excitement may be well managed by *hyoscyamus, anacardium, stramonium, valerianate* of *ammonia* and *passiflora.* The absorption of the exudate will call for *kali iodid, arsenicum iodid, baryta iodid, strontium iodid, silicea* and *sulphur.* Such remedies as *causticum, kali phos., picrate of zinc, strychnia phos.* and *argentum nitricum,* which we use in all degenerations of nerve tissue, may be employed in accordance with their indications as we approach the chronic stage.

The *mechanical treatment* of the paralyzed parts during the chronic stage will consider the application of electricity, massage and the systematic use of the arm and limb as much as possible. Faradism, to my mind, is still our best form of electricity for this purpose. Massage should be given by those who are trained for such service. Exercise of the affected parts is best performed by insisting upon the patient's gradually resuming the normal functions as much as possible. We may accomplish this by rigid insistence.

CEREBRAL SOFTENING.

The term cerebral softening is an unfortunate expression as it too frequently refers to the mental imperfection attending dementia. It properly means an acute softening which follows cerebral thrombosis or embolism. The softening and degeneration of the cerebral tissue are the results of the arterial occlusion, and lack of nourishment. The diminished blood pressure and the collapse of the arterial vessels favor a restriction of the venous circulation and, therefore, encourages venous stasis which augments the cerebral degeneration. The disease is characterized by a sudden apoplectic attack which simulates cerebral hemorrhage in its acute form. The subsequent symptoms partake of the character of chronic hemiplegia.

Etiology.—Embolism and thrombosis are more frequently experienced in early adult life. The predisposing influences of embolism are endocarditis, infectious fevers, diseases of the blood, and pregnancy; it is, therefore, a common disease in women. Thrombosis comes from the diseases which have a tendency to degenerate the arterial wall. Syphilis, lead poisoning, gout and fatty metamorphosis are the best known causes.

Pathology.—The pathologic process following either embolism or thrombosis develops rapidly, hence the term acute softening. At first anemia exists because the arterial circulation is cut off. This is more pronounced in the deeper white structure where there is less vascularity. On account of the venous constriction and punctate hemorrhages in the involved area, this will later show a sign of color and this is defined as "red softening." This form of infiltration is more pronounced in the cortex. Later, as the process of degeneration continues, a yellow softening is observed. Those conditions represent the stages of tissue metamorphosis and may vary to a considerable extent.

Soon after the arterial obstruction takes place, the nerve cells undergo degeneration. Granular formations displace the neuroglia and fiber formations until a general shrinkage of the involved part is noticed. Calcareous degeneration, cicatrization and even cysts are in evidence. If infectious germs are present local encephalitis or cerebral abscess may develop. *Thrombosis* is associated with arteritis and extensive atheromatous conditions. It seems to affect the larger blood vessels at the base of the brain. *Embolism* is associated most frequently with endocarditis, and the embolus lodges generally at the branching of the anterior or the middle cerebral arteries. Thrombosis and embolism may exist together and typical cerebral hemorrhage may attend either.

Symptoms.—The first observed symptoms of cerebral softening depend upon the character and location of the obstruction. Usually *embolism* presents a sudden onset and is quickly followed by hemiplegia. Coma, not so pronounced as in cerebral hemorrhage, is present and sometimes convulsions of the Jacksonian type are recognized. The other symptoms simulate those of cerebral hemorrhage, but are not so severe or lasting. Much depends upon the size of the clot and the artery affected. The occlusion of a small vessel may cause slowly developing symptoms, slight loss of consciousness and little hemiplegia.

In *thrombotic occlusions* the premonitory symptoms are more in evidence, and the subsequent symptoms are not so marked. The *onset* is not sudden; aphasia, numbness, drowsiness, vertigo and many other indefinite symptoms are experienced. Complete coma rarely exists and the hemiplegia is transient or slight. There are, however, cases in which the thrombosis presents all the extreme symptoms of cerebral hemorrhage, particularly when the middle cerebral artery is the seat of the occlusion. The hemiplegia in either embolism or thrombosis is not so extreme, nor does it run so persistent a course as in cerebral hemorrhage. *Monoplegia* alone is a common occurrence. We must keep constantly in mind the fact that when large vessels are involved the symptoms may be as severe and lasting as those of cerebral hemorrhage. If the typical symptoms continue for several weeks, we may expect a permanent hemiplegia. When the vessels of the basilar portion of the brain are occluded, the features of bulbar paralysis may appear. The palsy at first is flaccid, but later rigidity, exaggeration of reflexes and contractions may follow.

Diagnosis.—To properly differentiate cerebral hemorrhage and acute softening, from embolism or thrombosis, is no easy task. Hemorrhages may appear between the ages of thirty and sixty, while acute softening may appear earlier or later in life. *Endocarditis,* rheumatism or acute infection developing in early life predispose to embolism. If the symptoms occur very late in life they are presumptive of *thrombosis.* In *hemorrhage* there is generally a history of previous or hereditary arterial diseases and in acute softening we look for the history of syphilis, plumbism or alcoholism. In hemorrhage the onset is more sudden, the coma and paralysis coming on at the same time. In acute softening the onset is more gradual (particularly in thrombosis) and the coma is generally transitory. In hemorrhage the arterial tension is high, while in thrombosis it is low and the heart is weak. Hemorrhage almost always follows excitement, shock or violent effort; thrombosis is liable to occur during sleep or from no apparent cause. In hemorrhage rigidity is an early sign; in acute softening there are often limited convulsions. The pulse in hemorrhage is slow and full and the breathing is stertorous. In acute softening (particularly from a thrombus) the pulse is weak, soft and rapid. The hemiplegia following hemorrhage is more extreme and there is a greater tendency to later development of athetosis and choreic movements.

Prognosis.—The recovery from the early attack in embolism may be expected more than in hemorrhage or thrombosis. It depends much upon the cause, the condition of the patient and the degree of degeneration. Infectious diseases are liable to produce infectious encephalitis or abscess, which are, of course, dangerous. Advanced age is unfavorable. Recurrent convulsions and persistent temperature should arouse our fears. The chronic stage of hemiplegia will call for the same prognosis as in hemorrhage.

Treatment.—In the treatment we must consider the arterial diseases and the other predispositions to the occlusion of these vessels. *During the attack* the same care which is demanded in cerebral hemorrhage must be instituted. The patient must be kept quiet and in a semi-recumbent position. The *bowels* must be kept free in their action; the *kidneys* should be encouraged to a proper elimination and the *heart* must be effectually sustained. In thrombosis particularly we must stimulate the weak heart and so far as possible prevent the arterial excitement. *Digitalis, strychnia, strophanthus, adonis, convallaria, nitroglycerin* and *cactus* will here be useful. The *diet* must be limited for some time to liquids. In embolism we must particularly watch the heart lest any undue excitement shall thrust the embolus farther along or distend the artery to the point of rupture.

Kali iodid, arsenicum iodid, mercurius iodid and remedies of this character will naturally be suggested. Chronic hemiplegia will call for the same care as demanded under that chapter.

APHASIA.

This term refers to a speech defect which is due to cerebral disease. According to the disturbance of the cortical speech centers we have various imperfections of speech. These are defined as auditory aphasia or word-deafness, visual aphasia or word-blindness, motor aphasia or aphemia and graphic motor aphasia or agraphia. When the connecting fibers are disturbed a connecting aphasia may exist and in this way more than one center may be affected, giving rise to combined aphasias. As all of the speech centers are intimately related, we are liable to discover a combined aphasia in every speech perversion. These conditions also vary in different patients suffering from the same disease. With some the storage of word pictures is more perfect than with others, and the mental capacity also has a marked influence in overcoming these defects. The majority of speech defects occur in conjunction with hemiplegia. These are marked in the early stages of this disease but they gradually disappear in case of improvement.

Auditory Aphasia.—Word-deafness occurs when the lesion is located in the posterior part of the first and second temporal convolutions of the cortex. This region is the center for all sounds. Patients suffering with this form of aphasia do not appreciate the meaning of words when the auditory apparatus conducts them to the cortex. Music may be heard but not appreciated. When the auditory word memories are impaired the emission of vocalized words is imperfect. Thus the speech is reduced to a jargon, new words are often coined and syllables are repeated without any reason. The hand writing of such patients is also imperfect and the wrong words are used, while there is often a confusing repetition of words.

Motor-Aphasia or Aphemia.—The lesion which causes this form of aphasia is located in the foot of the third left frontal convolution and may extend into the foot of the ascending frontal, interfering, therefore, with the motor control of the lips and tongue. In right-handed people this lesion is on the left side of the brain and in left-handed people it is on the right side. Voluntary speech is impossible and the patient is not able to repeat words when he hears or reads them. Combined motor aphasia is usually present in this defect, and thus associate centers, particularly of vision and hearing, are affected. Agraphia is usually present in proportion to the motor aphasia.

Visual Aphasia.—A lesion located in the angular gyrus of the left side is the cause of visual aphasia or word-blindness. If the lesion reaches deep enough to involve the optic radiations from the basal ganglia to the cortex, hemianopsia may be added. Patients afflicted with this lesion are able to see written words but they convey no meaning to their center of visual appreciation. Spoken language is understood and the patient may repeat what he has heard. "When familiar objects are not recognized mind-blindness is present and the lesion is probably bilateral."

Graphic-Motor Aphasia.—The lesion which causes this form of aphasia is not fully understood. Wernicke claimed that the "act of writing is but the act of tracing the visual word memories," thus a perversion of this faculty will induce this defect. The theory that a graphic-motor center is found in the foot of the second frontal convolution adjoining the centers for the hand would be more substantiating but this has not been fully established. Inasmuch as agraphia is common in motor aphasia it is highly probable that this perversion of speech has an intimate relation to disturbances in the motor speech-center.

CEREBRAL PALSIES OF CHILDREN.

These occur as hemiplegias, diplegias or double hemiplegias and paraplegias. The *hemiplegias* differ only slightly from the ordinary hemiplegia as the result of cerebral hemorrhage, embolism or thrombosis. The *diplegias* or birth palsies are congenital double hemiplegias occurring usually as the result of injuries at the time of birth. Convulsions or prolonged asphyxia after birth may be overlooked as significant of cerebral trouble, and only when the child fails to use the limbs is the true condition recognized. The convulsions may persist or symptoms of epilepsy may appear and finally mental impairment is plainly evident. *Cerebral paraplegia* or *Little's Disease* is a rare form of this class of diseases. The paralysis is most marked in the legs, though the upper extremities are affected in extreme cases. There is no mental disturbance, and in the mild cases as the child grows older the paralysis may improve. The rigidity, contractures and exaggerated reflexes are liable to continue and these may be so extreme that the child's legs are crossed or flexed to such an extent that they are useless. In all of these cases spasticity and rigidity of muscles are characteristic.

Etiology.—The majority of these cases are congenital or occur during the first two or three years of life. Injuries to the mother during pregnancy, heredity and emotional conditions have some predisposing influences. Prolonged and difficult labor and mechanical delivery are probable causes. After birth any infection or injury which leads to intracranial hemorrhage are factors. Diseases of childhood and particularly cerebrospinal meningitis, may induce these diseases.

Pathology.—The pathologic conditions observed in these cerebral palsies are due to hemorrhage, embolism, thrombosis or a congenital defect in cerebral formation or nutrition. Another morbid cause is termed porencephalus, which means a cavity or depression in the hemispheres as the result of congenital atrophy or lack of proper development. This cavity generally communicates with the lateral ventricles. Meningeal hemorrhage, diffuse sclerosis or inflammation of the cortex are also occasional causes.

Symptoms.—*Convulsions* followed by fever generally announce the onset of this disease. *Paralysis* is the next symptom and it invariably is hemiplegic or diplegic. Paraplegia is a possibility, but is rarely observed. There may be temporary improvement after a time, but the affected parts do not properly develop; in extreme cases *atrophy* is marked. *Rigidity* soon follows and contraction of the flexors and adductors leads to deformities of various kinds. *Talipes* equino-varus and equino-valgus are most pronounced, but choreic, athetoid and associated movements are also common. The *hands* and fingers are flexed, tremors are present and ataxic movements may exist. *Mental weakness,* even to the point of feeble-mindedness, or idiocy, is apparent. *Epilepsy* is a frequent sequela. The *stigmata* known as the microcephalic or macrocephalic skull, together with facial asymmetry, imperfectly developed teeth, the taurus palatinus, etc., are often present. Taken as a whole, the child's mental and physical development is decidedly imperfect. There are some chances for improvement when the child passes puberty, but the chronic conditions defined above are inclined to continue indefinitely.

Diagnosis.—The characteristic physical and mental symptoms of the early and chronic stages should guide us in reaching a perfect diagnosis.

Prognosis.—The child may live to middle age or later, dependent, of course, upon the extent of the hemorrhagic cause and the imperfect development. A cure may be rarely looked for.

Treatment.—Since it is only in the chronic stage that the physician is called upon for treatment, *orthopedic surgery* or mechanical measures are about all we can suggest. In some cases *massage* and *electricity* may be called into use. If the limbs show any signs of positive improvement under these measures, we may be encouraged to try gymnastics and in every way possible cultivate the normal activities of the wasting limbs. The mental defects, in some cases, will be helped as the child grows older by educational measures and by remedies which aid the restoration of cell protoplasm, such as *phosphoric acid, ferrum phos., calcarea carb., calcarea phos., kali phos.* and *zincum phos.*

TUMORS OF THE BRAIN.

Tumors of the cerebral tissue include a variety of abnormal formations. New growths similar in structure to those found in other parts of the body and neoplasms peculiar alone to the brain may be recognized. Glioma, tubercle, gumma and sarcomatous formations are the most common. Myxoma, carcinoma, fibroma, lipoma, neuroma and vascular tumors may occasionally be found. New growths developing in the meninges and cranial walls cause identical symptoms and may be classed with brain tumors on this account. As a usual thing brain tumors are rare in comparison to other organic diseases of the nervous system.

Etiology.—Males are afflicted more than females, and all ages are susceptible; they rarely develop, however, after the fiftieth year. Tumors occurring during childhood are more liable to be tuberculous; gliomas and sarcomas develop occasionally at this period. Gummas, gliomas and sarcomas are more common after the twentieth year. Heredity seems to have slight influence and injuries are rare exciting causes. Metastasis may be the means of transportation from other parts.

Pathology.—The following facts are given by Dana: *Tubercle* is the form of tumor most frequently presented for pathologic study. It occurs as a single or multiple growth. It is irregularly round and varies in size from a pea to a hen's egg or it may be even larger; in number it varies from one to twenty or forty. The external appearance is a grayish yellow and internally it is yellow in color and cheesy in consistency. While the tubercle itself is not vascular, it is often surrounded by an area of tuberculous infiltration which may show inflammatory activity. The base of the brain is the favorite seat of tuberculous formation, though the distribution may be along the large cerebral or cerebellar vessels; the parietal convexity and the brain meninges may thus be involved. The tuberculous development is always due to the tubercle bacillus which has been transported by the blood vessels. The microscope will reveal the cell formation and the tissue degeneration characteristic of tuberculous growths. The majority of cases occur before the twentieth year.

Gummas or *syphilomas* point to a syphilitic meningitis or cerebral syphilis and their common location is at the base of the brain, or upon the surface of the hemispheres. The cerebellum or ganglia are rarely affected. They are irregular in shape, often multiple and are found on the meninges or large blood vessels. They are made up of small round cells, spindle cells and degenerated nerve tissue which appear as a cheesy mass in the center. Fibrous tissue increase, endarteritis and periarteritis of the peripheral blood vessels complete the pathologic picture.

Glioma is a development of the neuroglia tissue which normally acts as a padding for the nerve cells. It has its origin in the white matter, may attain a large size and is quite universally distributed in the cerebrum. It has a reddish or vascular color and, in consistency, it is soft like normal brain structure. It generally develops rapidly and displays a decided tendency toward infiltration and destruction of surrounding tissue. It is composed of glia cells and their fibrous prolongations. In the center of the tumor we may find the products of fatty degeneration which may become liquefied and develop into cysts. Sometimes the mucous degeneration leads to the formation of a myxoglioma or a fibroglioma when fibrous tissue is added.

Sarcomas are of frequent occurrence. They are of primary or secondary origin, their development is rapid and the tumor may attain a large size. Various shapes of the growth are recognized and, while they may be multiple, as a rule they are single and large. Frequently they are encapsulated. They are usually outgrowths of the dura or periosteum, and are located most commonly on the basilar surface. Sarcomas are made up of small round cells, spindle cells and other cells; fibrous tissue may be excessive and, though containing numerous blood vessels, they are not usually vascular. Modifications of the typical form exist in accordance with the amount of connective tissue increase and the process of degeneration. Fibrosarcoma, myxosarcoma, gliomasarcoma, cyst and other formations will be recognized by their histologic structure.

Cysts are sometimes found in the brain. Abscess encysted, hemorrhagic exudates, the central softenings of sarcomas or gliomas, and dermoid developments will give rise to this form of fluctuating tumors. Cysts caused by the cysticercus cellulosae or the echinococcus, may exist, but they are rare in this country. Cysts in the central portion of the brain may be found as the result of ventricular distention and, in rare instances, the pituitary body may suffer similar degeneration. All of these cystic developments are of comparatively slow growth.

Intracranial aneurysm will often present symptoms indicative of brain tumors. Miliary aneurysms are usually multiple and appear as slight dilations along the course of the vessel. Large aneurysms occur in later life as the result of arterial disease or, in exceptional cases, they may arise from traumatism. They are found most frequently at the base of the brain. The middle cerebrals, the basilar, internal carotids and anterior cerebrals may be the seat of the lesion. The characteristic symptoms are much the same as those of tumor at the base of the brain. Focal signs will appear when cranial nerves or other motor centers are involved. Optic neuritis is not usually present. The patients are sometimes conscious of pulsating sensations.

Other forms of tumors may be found in the brain tissue in rare cases. Fibroma, osteoma, angioma, neuroma and lipoma may be mentioned in this class. Carcinoma appears less frequently than any of them.

Symptoms.—In the study of symptoms pertaining to brain tumors, we shall find that many of these may be regarded as general, inasmuch as they do not have direct reference to the character and location of the growth. On the other hand there is a class of symptoms which directly refers to the location of the growth. The symptomatology then may be divided into general and focal symptoms.

General Symptoms.—*Headache* is an early and persistent symptom in nearly all brain tumors. It varies according to the location and development of the tumor, and while it is often localized in the vicinity of the growth, it may be so general as to resemble a crushing sensation. The *severity* of the pain is augmented when cerebral hyperemia or arterial tension are increased. In character it is paroxysmal and very intense. As the tumor becomes large in size and causes increased intracranial pressure or encroachment upon the membranes, the pain becomes unendurable and constant. Local tenderness and local elevation of temperature are rare symptoms. *Vomiting* invariably attends the development of the tumor and is often as persistent as the headache. It is distinguished from that of gastric origin inasmuch as there is no nausea. The vomitus is ejected from the stomach with great force and the act is described as projectile in character. These attacks are liable to subside for a time, only to recur with greater intensity. This symptom is associated with rapidly developing tumors like the syphilitic or tuberculous, and it is most pronounced when the cerebellum and corpora quadrigemina are involved by the new growth.

Vertigo is a common symptom which is most frequent when the frontal lobes or the cerebellum are affected. It may be slight and not complained of, or it may be paroxysmal and severe. Usually it is a constant disturbance. When the cerebellum is implicated it is liable to be severe and attended by forced movements.

Optic neuritis will be discovered in the majority of cases. It occurs most frequently in tumors of the cerebellum, midbrain and the basal ganglia. When the medulla is the seat of the tumor it is not so common, and it will not be so marked in the slowly developing growths. *Atrophy* of the optic nerve is the eventual termination of optic neuritis. At first it may be more marked in one nerve, but both are soon affected.

Mental impairment is another sequence of brain tumors. This may be simply a persistent hebetude or it may become a pronounced stupor or coma. In the early stages hebetude is most characteristic; the patient is dejected and indifferent, speaks slowly and shows a sluggish mental activity. The *respiration* is shallow, the pulse is slow and the condition soon becomes one of apathy which may merge into a comatose state. Tumors of the frontal lobes show these symptoms most frequently.

General convulsions are early signs in many cases. They simulate the epileptic attacks, but resemble more particularly the Jacksonian form. The attack, however, is less violent and abrupt, and the clonic stage is usually prolonged; the coma is not so pronounced and the seizures do not recur with pronounced regularity.

Focal Symptoms.—These vary according to the seat of pressure and the extent of anatomic destruction. Small tumors may give only slight symptoms of unilateral spasm, while larger ones cause *paralysis* of the affected nerves proportionate to the impairment of special nerve involvement. *Monoplegia,* hemiplegia and motor aphasias are characteristic *motor* symptoms, while hemianopsia and functional paraesthesia will be indicative of *sensory* disturbance. *Cranial nerve* impairment and the Jacksonian fit will be suggestive symptoms for study. The exact localizations must be made by the aid of our knowledge of the cerebral topography.

Prognosis.—The results of brain tumors point invariably to death. Tubercle and gumma offer some hope from treatment.

Treatment.—Tuberculous and syphilitic growths present an opportunity for help by means of internal remedies. These should be applied in accordance with the accepted theories of treatment in that class of cases. There is some claim for *arsenic* in sarcomatous tumors, but this and other remedies have not yet yielded sufficient results to warrant any extravagant claims for it. Symptomatic treatment will always give them some temporary relief in the majority of cases and the general conditions will need attention the same as in other diseases. *Opiates* and *anodynes* will be demanded for the relief of pain, and any measure which will improve the nutrition should be instituted.

Surgical procedures may be demanded when the tumor is clearly operable. Even the symptoms in inoperable tumors are somewhat relieved by opening the skull. The hard and slowly developing growths may be removed, providing the knife does not destroy vital tissues. Tubercle, fibroma and sarcoma have been removed with good results. Cerebellar, pons and medulla neoplasms will not warrant surgical interference. Cysts and abscesses may often be evacuated and their sacs removed with marked relief.

GENERAL PARESIS.

Paresis is a term which has been employed to cover various manifestations of nervous debility. "General paralysis of the insane" is a term which seems to exaggerate the idea of acute insanity with an attending paralysis, while "dementia paralytica" would emphasize the feature of dementia, of which a general muscular weakness is a sequence. The word paresis has been much misused, inasmuch as it is employed with reference to a general breakdown, or a loss of mental activity which borders upon insanity. We may regard it as a progressive disease of the brain which generally ends in dementia. There is associated with this a peculiar physical weakness which virtually terminates in a paralysis.

Etiology.—This disease is most common in men. *Heredity* does not play an important role in its history, though it is probably encouraged by an inherited nervous debility. *Excesses* no doubt constitute the most frequent causes and alcohol may be named first among these. Prolonged *mental exertion,* emotional excitement and excessive sexual indulgence have their influence in the disease. *Syphilis* is the most frequent cause. This disease has, therefore, a close relation with such forms of degeneration as tabes dorsalis. *Injuries,* exposure, sunstroke and the acute infections have some bearing upon the disease as irritating factors at least. If to these are added syphilis, together with mental strain and the other excesses, it will be quite natural to expect the development of paresis. *Middle life* furnishes the most cases, and it usually runs its course in two to five years and in some instances in less time.

Pathology.—Degeneration is observed in the brain cells as the result of syphilitic infection. The membranes and the arterial coats are thickened while there is considerable perivascular proliferation. The brain becomes atrophic and there is a tendency to increase cerebrospinal fluid. The pia mater is decidedly congested with thickening of this and the arachnoid and a general adherence to the cortex. Connective tissue increase attends the nerve cell degeneration. The cells and nuclei of the medulla are affected as well as the cortex. It is not uncommon to find the lateral tract sclerosis of tabes dorsalis as an attending complication.

Symptoms.—The *first stage* is characterized by excitement or irritation, and the *second stage* gives expression to the dementia and paralysis. These are subject to modifications in many instances and it is possible for the disease to exist without any clearly defined manifestations of the first stage. Thus *hypochondriasis* or melancholia may terminate gradually in dementia, and the physical infirmity will be finally recognized as the mental weakness develops. On the other hand the mental symptoms may be of minor import, and the disease will then show more of the motor perversion of a brain and cord disease.

In the early stages of the *excited type* the tendency to irritability is very marked. The patient shows unmistakable signs of *mental perverseness;* he is fretful, fault-finding and often quarrelsome. His mental activities are not up to the normal standard; he suffers with lapses of memory; he makes unexplainable mistakes in his work and his statements are often suspiciously extravagant. *Fatigue* becomes more and more pronounced, so that he is exhausted from slight exertion. Alternating with these periods of *depression* are periods of *mental exaltation.* He expresses extravagant opinions of his own ability in comparison with that of others. He may become so loquacious that his mental weakness becomes a subject of gossip, and he may possibly undertake large and unexpected business ventures. He may spend his money foolishly under the delusion that he is very rich or has the prospect of a prodigious income. In this stage the elements of grandeur and magnificence characterize his conversation and his actions.

During this period of exaltation tremors are gradually manifested. The hands, the face and the tongue show this tremulousness when voluntary action is undertaken. The *speech* is imperfect and the stuttering habit appears. The *knee-reflex* is exaggerated, the pupils are uneven and some signs of the Argyll-Robertson pupil are evident. In a small number of cases the knee-reflex is lost, suggesting the complication of a posterior sclerosis. The *muscular weakness* constantly increases and the patient notices that it is not possible to take very long walks or remain long at a time on the feet without suffering unusual exhaustion. The *bladder* and *sexual function* are weakened; *insomnia* is often disturbing and, on rare occasions, apoplectiform attacks or epileptic seizures are experienced.

After the first stages the patient usually is more calm in his actions, which naturally suggests an improvement. It will be noticed, however, that he is gradually drifting toward *dementia.* He is inclined to sleep, shows little interest in his surroundings, and, of course, is unable to work, since his memory fails him and he has not proper control of his voluntary actions. These perversions rapidly increase until it is no longer possible for him to conduct an intelligent conversation, and *impairment of motor power* is evident. He is inclined to sleep much, he becomes careless about his appearance, becomes slovenly in his habits, and, in many ways, manifests the fast approaching mental weakness. At this period the apoplectiform attacks may reappear, causing a temporary or permanent hemiplegia. The epileptic seizures may also return and with one complication after another the patient reaches a state in which delusions, delirium or the symptoms of imbecility supplant the normal mental activity.

When this stage is reached the patient becomes bedridden from the general paralysis and exhaustion. The muscular inefficiency may persistently increase until he is absolutely helpless, and in some instances the power of speech also disappears.

The case may assume a rapid course, in which death comes to his relief within a year, or it may possibly continue as a partial or complete dementia for many years.

The *hypochondriacal type* may begin with symptoms which simulate the ordinary neurasthenic case. In such instances the fine tremor, the exaggerated reflexes, the insomnia, the digestive disorders and all the peculiar phenomena which appear in the symptomatology of this disease may be recognized. The physical debility—which amounts to paralysis—and the mental features appear in the later developments.

In another type *dementia* is recognized as a primary symptom. It may start as a mere weakness of the mental activity and gradually assume the more violent phases of mental perversion and physical infirmity.

There is still another type in which exudative syphilis is more of a feature. In these the early attacks will be ushered in with eye palsies, hemiplegia, intense headache and convulsions. Improvement is obtained under antisyphilitic treatment, but the mental and physical infirmities follow as in the other varieties of the disease. A similar condition of mental and physical weakness may follow the prolonged use of alcohol, but it does not reveal the distinctive signs of paresis unless there is a complicating specific cause.

Diagnosis.—Neurasthenia, acute mania, epileptic dementia, paranoia, multiple sclerosis and the mental conditions which attend organic brain lesions should be differentiated from paralytic dementia. *Neurasthenia* closely resembles the prodromal stage. The fine vibratory tremor, the exaggerated knee reflex, the complex symptoms which greatly annoy the patient, the absence of mental impairment and the lack of any grave motor or mental symptoms are sufficient to distinguish this from the serious aspect of paralytic dementia. In chronic *alcoholism* the tremor, epileptiform crises and mental changes are symptoms which closely simulate paresis. The improvement which follows the withdrawal of alcohol will, however, establish a sufficient differentiation. It must be remembered that this condition may predispose to paralytic dementia.

Multiple sclerosis, though at times simulating this disease, nevertheless is differentiated by the marked intention tremor which is absent in paralytic dementia. Nystagmus is almost never present in the latter disease.

Prognosis and Treatment.—The prognosis is invariably unfavorable, death occurring within a few years. The *treatment* must include all the features of palliation described under the subject of neurasthenia and hysteria and a similar class of remedies will be demanded. Advanced cases certainly require institutional care. Antisyphilitic treatment is indicated when a specific history is obtained, even though it has been previously employed.

DISEASES OF UNCERTAIN PATHOLOGY.

EPILEPSY.

The term epilepsy has been subject to various interpretations, and it has been used to cover the definition of many peculiar expressions of disease. In early times its occurrence was even attributed to the wrath of mythological deities. Our knowledge of this disease is not yet perfect. Not every convulsion or epileptoid seizure should be classed as epileptic, and in every instance we should determine the pathologic condition before we make a diagnosis or promise a cure. It is probably a disease of the cerebral cortex and when there is excessive irritation of this part it is characterized by sudden and disorderly discharge of motor force. This may come from a morphologic cellular change in the cortex or it may be traced to a variety of pathologic causes which point to a neurosis of some kind. It may also be encouraged by physical or mental degeneracy.

We should look upon the *attack* or seizure of epilepsy *only as a symptom;* the disease may be anything which interferes with the mechanism of the motor energy stored up in the motor cells of the cortex. There are many forms of seizures which have been erroneously included under this term.

Etiology.—*Predisposing causes* have much to do with the development of epilepsy. *Neurotic heredity* is a factor to consider. Such diseases as *syphilis,* phthisis and inebriety are without doubt predisposing factors. *Consanguineous marriages* may accentuate a neurotic tendency which contributes to its development, and the emotional temperament should always be regarded as favorable to its occurrence. The *epileptic age* is between ten and twenty, the majority of cases occurring before the latter year. When the seizures appear after this year, organic brain disease, or some acquired cause like syphilis, alcoholism or plumbism may be suspected. *Idiopathic cases* may begin in infancy and are reported as late as the sixtieth year. The period of dentition may set up a reflex brain irritation which later may develop into a true epilepsy. Every convulsion in a child should be regarded as serious until we are satisfied that it is not epilepsy. This is always a progressive nervous disease. Sex does not seem to have a special influence.

Inciting causes must have behind them these predisposing defects—hereditary influences and degenerative neuroses. *Reflex irritations* are presumable causes, hence, the ocular and auditory irritations, gastric and intestinal disturbances, pelvic and genital diseases, dental irritations, and, in fact, any peripheral cause which abnormally excites the sensory tract may cause a seizure. In this class may be included those cases which seem to have a relation to childbirth or perverted menstruation. *Head injuries* must be regarded as common causes in producing focal epilepsy. *Toxic agents* are decidedly predisposing and we may often trace the cause of the disease directly to alcohol, tobacco, morphin, cocain and similar intoxicants. The toxemia may come from the intestinal tract or through the faulty elimination by the kidneys. *Infectious diseases,* particularly scarlatina and typhoid, are occasionally causative agents and in all probability other acute infections are contributory influences in a fair share of cases. *Syphilis* must always be recognized as a very common cause, either from the hereditary standpoint or as a factor in the cortex changes. Other inciting causes no doubt exist and we may reasonably presuppose that any prolonged or unusual irritation which spends its force upon the motor cortex may induce this disease.

Pathology.—The final pathology of epilepsy is yet to be written. That a cortex disturbance is the underlying cause there is no doubt. This may come from a degeneration of the motor cells, or it may arise from a proliferation or increase in the neuroglia tissue. Special areas of the cortex are usually affected and secondary changes like connective tissue increase and vascular perversions may contribute to the prolonged irritation. *Congenital defects,* acquired malformations, nutritional errors and traumatism may all conspire to depreciate the normal cortex function or produce a reflex irritation which favors the sudden and explosive discharge of motor force.

These anomalies pertain to the body as well as the brain, yet in the latter we may also occasionally find a localized perversion in the convolutional outline, which has some pathologic significance. *Chronic leptomeningitis,* cerebral congestions and minute hemorrhages no doubt have some relation to the direct pathology. *Meningo-encephalitis* and disseminated sclerosis in both the cortex and the ganglia must also be regarded as causes. While cranial asymmetry may predispose to convolutional imperfection and cell inefficiency, a focal irritation may induce the seizure, or a general cortex excitement may follow from reflex disturbance.

Symptoms.—The *attack* is generally preceded for a few hours or a few minutes by a peculiar feeling called the *aura.* This is a premonition that a seizure is about to take place and it may be defined as a malaise, an irritability or a sense of giddiness. In those instances in which it precedes the convulsive explosion by several days it is characterized by such motor disturbances as twitchings of the face or extremities, tremors and restlessness. Sensory paresthesiae, mental excitement or depression may characterize this premonitory stage. While the aura is usually an initial symptom, it may not always be present, and in some cases it may represent an aborted attack. Besides the motor and sensory aura a *psychic aura* has also been described. In this there is a peculiar accentuation of the mental faculties and this is marked either by a sense of depression or an irritable or emotional impulse. A *cerebral aura* may be defined as a sudden vertigo or pain in the head. We may also discover a *visceral aura* which may relate to sensory or motor excitement of different viscera. Under this head we shall recognize such complaints as precordial pain, cardiac palpitation, laryngeal spasm, gastralgia, colic and incontinence of the vesical sphincter.

The *convulsion* or epileptic fit varies from the minor attacks, which are not always recognized, to the major form with the classical symptoms. In a typical case of epilepsy, the convulsion appears suddenly, a *shrill cry* is frequently uttered, *unconsciousness* immediately follows, and the patient falls in the fit. Three distinct conditions then define the seizure—the periods of *tonic* and *clonic* convulsion, followed by a period of *stertor.* In the *tonic stage* the most distressing features of the fit occur. It is on account of this tonic spasm that the patient falls so violently and may be injured; for the same reason we may account for the sudden fixation of the laryngeal muscles which causes the *peculiar cry* or an expiratory guttural noise. The whole body is in a state of rigidity. The *tongue* often gets between the teeth during this stage of the spasm and, hence, bleeding at the mouth is often noticed. *Vascular lesions* in the skin, mucous membranes or conjunctiva may also take place at this time, and it is possible for the brain and internal viscera to be affected in a similar way. The tonic stage usually lasts only a minute or two, and in some cases only fifteen or twenty seconds.

The *clonic period* slowly supplants the tonic spasm and is evidenced by the gradual jerky movements of the face and limbs as muscular contractions begin to take place. The slight agitation of the muscles soon develops into the grosser and more vigorous muscular movements, in which the patient may be thrown from bed, while fracture of a limb or other injuries may occur. The *violent contractions* of the muscles of mastication and the grinding of the jaws may severely injure the tongue, and accentuate other facial symptoms of the rigor stage. The *rapid clonic* movements after a short time gradually give way to less frequent contractions, but their violence may persist for some time. As the *respiration* is still in a state of partial suspension the asphyxia and cyanosis continue. The *heart action* is intense, the arterial tension is high, the temperature may be raised from one-half of a degree to one degree, and the body may be covered with perspiration. This stage terminates in one to five minutes and usually ends by a gradual or sudden condition of relaxation. Involuntary urination or defecation may occur as the result of sphincter relaxation.

The *period of stertor* is characterized by the complete unconsciousness and the deep stertorous breathing. This may be so profound that recovery of the conscious state never takes place, though this is a rare occurrence. In a very few minutes the patient opens the eyes, recognizes people, gains partial control of his motor power but, aside from a slight exhaustion, has no remembrance of the fit. Following this period the patient usually passes into a deep sleep. The mind does not attain its normal function for several hours and a condition of exhaustion may exist for a longer time.

There may be *many modifications* in the character of the epileptic convulsion and the duration of the different periods, but the features as outlined in the three stages just defined will accurately characterize the seizures. Sometimes the attacks occur at night and these forms of *nocturnal epilepsy* may not be discovered until they become excessively severe or possibly occur in the evening or morning. Many of the seizures take place with decided regularity and, therefore, may be guarded against, particularly when there is a pronounced aura. *Other attacks* occur at such irregular intervals that it is impossible to anticipate them, so that these patients invariably suffer some injury from the fall at the beginning of the attack. A condition, known as *status epilepticus,* may be a terminal condition of epilepsy. It is essentially a prolonged state of epilepsy. It is followed by a period of profound prostration or collapse, but frequently death may take place in the convulsion.

Symptoms of the Minor Attacks.—The incomplete attacks, known as the *petit mal,* often take the place of a typical complete attack or may be a distinct type. In many instances they are not recognized by either the physician or the patient. They are characterized by a sudden and momentary loss of consciousness. The patient may nod his head, drop something from his hand, may fall or forget what he is talking about. During this time the features may become fixed, the face may suddenly blanch and a vacant look may be detected in the half-open eyes. Further than this there may be no evidence which suggests epilepsy and the patient is frequently unconscious of the interruption of his faculties.

When the *aura* is *present* it may not always be appreciated. Many of the minor seizures are known as *vertiginous attacks.* In these the patient suddenly loses consciousness and nearly falls. The facial muscles twitch slightly, a quivering is noticed about the eyes and mouth, the limbs are moved with difficulty and the head may be slightly drawn to one side. Usually recovery takes place almost instantly and only a temporary sense of exhaustion follows. In rare instances a form of *procursive epilepsy* is experienced. In this the attack is evinced by sudden forced movements. The patient runs a short distance, turns around or makes automatic movements. *Symptoms of the psychic form* are characterized by outbursts of maniacal excitement and during these the patient may do injury to himself or to others. These states of excitement may terminate suddenly with a condition of collapse or they may be protracted to such an extent as to simulate true insanity. Under such an influence homicidal acts may be committed, obscene language may be indulged in, or a pyromaniac habit may be established. Again, the patient may be subject to sudden and violent automatic actions, while in the semi-unconscious state of the partial fit. In other instances the *somnambulistic state* may prevail, and while in this the patient may walk about as if asleep. A *psychic epileptic equivalent* has been described as a form of epilepsy which comes on without any preliminary attack. In other instances hysteria may complicate or mask the epileptic factor to the extent that hysteroid convulsions are recognized. In these the patient performs many coördinate acts the same as in true hysteria.

Jacksonian or partial epilepsy is characterized by a convulsion which affects only a group of muscles and is not usually accompanied by loss of consciousness. The irritating cause is always a focal lesion and may be caused by a tumor, injury, or an inflammatory exudate. The fit may at some period of the disorder have all the features of a grand mal attack. *Post-paroxysmal phenomena* are usually recognized as transitory conditions which follow the shock of a seizure. These may be characterized by special sense perversions, reflex excitement or they may partake of motor disturbances, such as tremors, speech impairment or some form of temporary paresis. Much depends upon the intensity of the attack, but the patient usually sleeps immediately after the convulsion and, with the exception of the involuntary loss of urine and the stupor, it seems like a normal sleep. The *temperature* is usually elevated a degree or more for a short time and the patient may complain of headache, while vomiting is liable to be a distressing symptom. The *urine* shows an increase of earthy phosphates and its toxicity is probably enhanced. The most unfavorable sign at this period is the evidence of mental deterioration which is gradually augmented by the repeated attacks, and in some instances it may be followed by dementia.

The *general health* does not suffer much impairment and, aside from the digestive disturbances which may occur from overeating and the remedies employed, we shall notice little out of the ordinary except the increasing mental incapacity. The physical imperfections, among those who are born to epilepsy, have been called *stigmata.* These are defined as the short stature, cranial deformities, bad teeth, high palatal arch, facial asymmetry, imperfectly developed ears, atrophic genitals, together with the signs of many physiologic deteriorations and psychic stigmata. While all of these may be contributing influences they do not necessarily have any direct relation to the prevalence of convulsions.

Diagnosis.—The diagnosis of a typical attack of epilepsy is not a difficult matter, but the recognition of many of the incomplete attacks will often tax the observation of the best physicians. This is particularly true of the nocturnal attacks. Careful differentiation will require close study in many instances, for the epileptoid seizures, which arise from functional disturbances and toxic conditions, often suggest the true attack. For this reason we must closely watch those convulsions which arise from intestinal and other reflex disturbances, inasmuch as the removal of their cause will eliminate the fits. It is claimed, however, by the best experts that those convulsions from reflex causes may induce a real epilepsy in those who are predisposed. We must remember also that epilepsy may appear as the result of more than one cause and, therefore, it is necessary to watch every contributing irritation lest this become the means of exciting a latent predisposition or of contributing to the developing pathology. To be sure of our diagnosis the periodicity of the attacks must be established. *Hysterical convulsions* will often simulate epilepsy, but these never have the true loss of consciousness, while their distinctive symptoms—as enumerated under that subject—are generally well defined. *Tumors* of the *brain, apoplexy* and some of the vertigoes may resemble epilepsy, but other attending symptoms usually remove the doubt.

Prognosis.—The danger in epilepsy does not exist in the single attack. The seizure is not, in itself, the worst feature, but its debilitating effect upon the brain is liable to terminate in dementia or at least in impairment of the mental function. The prognosis is, therefore, unfavorable, and that is why so few physicians of standing offer any encouragement from treatment. There are a class of epileptoid seizures—dependent entirely upon reflex causes—which we may hope to relieve, but these are not in reality epilepsy. It is safe to say also that a cure may be expected whenever the exciting cause is completely removed, while a condition of degeneracy is always unfavorable. The prognosis in petit mal is often as unfavorable as in grand mal and the psychic form is still worse. Status epilepticus is always an unfavorable condition and any organic cause will make the outcome less hopeful.

Treatment.—The treatment of epilepsy is a discouraging undertaking for the physician, for neither immediate improvement nor a permanent cure are to be expected. The dissatisfied patient is bound, sooner or later, to vacillate between physicians and surgeons, or to supplement one patent remedy for another at the earnest solicitation of interested or meddling friends. Thus it is rare that any practician has a fair opportunity to try any remedy or a particular line of treatment. Surgery has, to my mind, done more to discourage medical treatment than anything else. The laity has imbibed the idea that the disease is something to be removed by operating upon the brain, and for this impression an unscrupulous or ambitious surgeon is often responsible. That surgery is indicated in some focal lesions like Jacksonian epilepsy or in the case of cranial depression, I have no doubt. In the average case, however, the irritation created by the post operative cicatricial increase, overbalances the temporary improvement which is invariably apparent soon after the operation.

In the line of internal medication *bromids* are used most universally, but it is also questionable if a positive cure was ever accomplished by this agency. That bromids suppress the seizures and prolong the interparoxysmal state there is no doubt, but no more than palliation should be claimed for this agency. On the other hand, the danger of bromidism is always to be feared. There are several forms of bromids—*potassium, sodium, strontium* and *ammonium*—which have been used with varying success, but the one most frequently employed is the bromid of soda. This is the most popular inasmuch as it does not disturb the stomach so much as the others, if it is taken with a sufficient amount of water. The *bromid* of *potash* probably has a better sedative effect, but it is more liable to create stomach irritation and other unpleasant effects. The bromid of gold and hydrobromic acid have been also recommended, while many advise the combination of ammonium, potassium and sodium bromid.

As there are opinions for and against the use of bromids, so, too, there are various opinions regarding their method of administration. Some hold to the theory that it is best to give a mild dose (gr. 10-15) three or four times daily, and gradually increase the dosage to the maximum of endurance. Others argue that it is best to start with a larger dose (gr. 30-60) and increase gradually. Some claim that "if sixty grains daily will not correct the seizures, then nothing will." Others, however, hold that thorough bromidization is absolutely necessary. It is claimed also that the omission of salt from the diet will favor its action by aiding the absorption of the bromid, and thus smaller doses may be used. Church advises the employment of the largest dose after the evening meal, when the seizures are nocturnal, and at bedtime when they occur in the morning. The danger of bromidization may be limited by increasing the physical exercise, taking salt baths and using mineral acids and alkaline waters. In my own practice, when I have been discouraged enough to resort to bromids, I have given the following compound tablet: Sodium bromid and potassium bromid (each grs. 3), cannabis indica and hyoscyamus (each m. 5), in slightly increasing doses, four times daily. It has, in my experience, suppressed the seizures, with less danger of bromidism than is experienced when larger doses are given.

The use of *sedatives* has been praised by some, but with these we only gain—in another way—temporary respite from the horrors of the disease. For this such remedies as phenacetin, antipyrin, trional, chloral hydrate and chloralamid have been advised. In status epilepticus, chloral, or any of these remedies, may be given in large doses by enemata. The nitrite of amyl pearls may be of adjuvant use in giving temporary relief to some cases or more particularly in warding off attacks during the early stages. Inhalations of chloroform and ether have been used with a similar object.

Many *specific remedies* have been employed, but the majority have been abandoned after a thorough trial. *Belladonna,* on account of its power of dilating capillaries, has been used with the idea of giving relief to cerebral congestion and irritation and the same is true of *nitroglycerin.* The reaction which follows the prolonged use of these has discouraged their extensive employment. *Digitalis* has also been used with the idea of stimulating the heart and thus correcting the predisposing influence of a weak circulation. *Strychnia* and *opium* have been employed from an opposite standpoint with the idea of blood limitation in the affected part. Neurasthenic remedies have also been employed, the aim being to limit the nervous excitement which certainly must favor the explosive discharge of motor force.

The regulation of the *diet* is a most important consideration. Nitrogenous food should be limited. Furthermore, it will be necessary to limit even the vegetable diet inasmuch as these patients are inclined to ravenous appetite. The gastric and intestinal irritations, which may follow from overeating, will favor the seizures, the same as worms in the case of children. Observations relative to the general health are of great importance here as in other diseases, for as we perfect the equation of physical resistance we put our patient into the best position to resist the disease and respond to our remedies.

Homeopathic remedies are of value in the treatment of epilepsy, but, as I have previously said, I have my doubts about any remedy curing this disease, and this opinion is expressed only after an earnest effort in this respect covering many years of practice. Any remedy or any treatment which will correct the pathologic condition will cure the disease, and if symptoms are a guide to that end, then our study should be continued in this line for some time. The value of the homeopathic remedy often consists in the relief of irritations which favor the pathologic development and the periodic explosion of motor force. If they accomplish nothing more than this they are certainly of service in the treatment of this disease. *Enanthe crocata, verbena hastata* and *solanum carolinense* are probably the most popular remedies used by practicians of our school. The first bears a symptomatology closely allied to idiopathic epilepsy, but it has failed me in all its potencies, and I have seen only temporary relief from the use of the tincture. *Verbena hastata* often helps a certain class of cases in which the reflex features are pronounced, while *solanum carolinense,* to my mind, is the best remedy of the three. This I use in the tincture (m. 10-30) four times daily. *Avena sativa* has received favorable mention for cases in which neurasthenic symptoms are marked. *Aurum mur.* may pertain to tissue changes in the cortex and for that reason it is often regarded as a remedy for consideration. This, together with the *iodids, mercurius, argentum nitricum* and *nitric acid* will naturally be indicated when there is an underlying specific cause. *Hyoscyamus, stramonium, cannabis indica* and *belladonna* will relieve many of the symptoms of cerebral irritation which certainly favor the discharge of motor force. Therefore, it would seem to be a correct method of practice to prescribe such remedies when they are symptomatically demanded. *Anacardium* should not be overlooked when mental excitement is a predominating symptom.

A certain class of remedies which have a direct relation to the neurasthenic and hysteric conditions will require careful examination in the treatment of many cases. Thus *ignatia, strychnia, zinc phos., zinc pic., asafetida, valerian* and *kali phos.* relate directly to the causes which at least favor the epileptic attack. The spastic or choreic condition of the muscles will frequently demonstrate a state of nerve excitement which is relieved by such remedies as *cuprum metallicum, agaricin, cicuta virosa, plumbum* and *amyl nitrite.* And so we might enumerate many other homeopathic remedies which, if they do not cure epilepsy, may at least help us to correct the severity of its symptomatology.

HYSTERIA.

The word hysteria has been applied to such a variety of nervous diseases that it has become almost a misnomer in medical practice. As it gives rise to so many peculiar mental manifestations we may consider it a psychic disturbance. In addition to this, trophic and vasomotor disorders are sufficiently prominent to class the disease among the neuroses. It is often characterized by crises of an emotional or convulsive nature and the interparoxysmal state will be recognized by various stigmata.

Etiology.—*Heredity* plays an important part in the history of this disease, as many cases can be traced back to a neuropathic family trait. This rule, of course, applies more particularly to children than to adult patients. Rheumatism, gout and tuberculosis also frequently predispose and a faulty early training is likewise a contributing factor. The *age of puberty* and early adult life furnish the greater number of cases, though this rule is often modified by temperament and circumstances. The female is supposed to be affected most frequently, though the male is by no means exempt.

The *inciting* cause of most frequent occurrence is emotional disturbance, though fright, grief and worry are often incidental causes. Traumatism is also a factor of importance among males if the mental shock is intense at the same time. Infectious diseases, cachectic conditions, intoxications by lead, alcohol, mercury, cocain, morphin or tobacco, associate nervous and mental diseases and overwork as well are part of the direct causes. Sexual excess may induce many cases, while a large class of patients develop the disease through imitation.

Symptoms.—The true symptomatology of hysteria is essentially an interpretation of the stigmata. These partake of a sensory, motor and psychic character. The *sensory symptoms* include both the hyperesthesias and anesthesias. *Hyperesthesia* may be detected in various forms. Extreme sensitiveness may exist, either in a circumscribed area or in a general distribution which seems to be a part of the extreme nervous excitement. The circumscribed form is usually confined to small superficial zones on the breasts, in the groins, along the spine, over the ovaries or in the epigastric area. A *neuralgic pain* may supplant the hyperesthesia. The pain may sometimes be confined to a localized headache, either on the vertex or over the temples, and its extreme exacerbations may simulate meningitis. *Intercostal pains,* facial neuralgia and spinal irritation may alike represent the hyperesthetic phase of the disease Pressure upon the hyperesthetic areas is liable to excite various kinds of paroxysms and, hence these spots are termed hysterogenic zones.

Anesthesia is a more pronounced feature of hysteria than hyperesthesia. This may be represented in various degrees in different stages of the disease. The loss of sensibility may be partial or complete, it may vary in extent and distribution, it may also involve superficial structures like the skin and mucous membranes or it may invade the deeper tissues like the muscles, ligaments, bones and nerve trunks.

The *distribution* of *hysterical anesthesias* is somewhat peculiar. The most common form is that of hemianesthesia which involves the half of the body, generally including the superficial mucous membrane of that side. It may also implicate the special senses. The *segmental* distribution of anesthesia is the next in frequency and this will be recognized by the loss of sensibility in one arm, a leg, a part of the face or head, or it may be defined by a band-like area of anesthesia which girdles the body. *Disseminated anesthesia* is a rare form in which the loss of sensibility is distributed in patches about the body. In all of these varieties the perversion of the pain sense is most pronounced, the tactile and thermic sense being usually less affected. The *peculiarities* of the hysterical anesthesias are quite marked. The loss of sensation does not conform necessarily to the anatomic distribution of the cutaneous nerves. The majority of the reflexes, with the exception of the palpebral and pharyngeal, are retained or rarely modified, the knee jerk and ankle clonus being inclined to exaggeration rather than a decrease. The anesthetic zones are changeable, often being modified, aggravated or increased in size by an hysterical attack; the loss of sensation is rarely recognized by the patient until attention has been drawn to this fact. The anesthesia usually disappears during sleep and when the attention is diverted.

The *motor stigmata* of hysteria are important phases of the disease and we may observe marked errors of movement which vary according to the extent of the disease and the susceptibility of the patient. *Retarded* movements are very common, the degree of perversion depending—to a large extent—upon the amount of anesthesia. *Incoördinate* movements are also present in hysteria proportionate to the anesthesia and the loss of muscle sense, and this incoördination disappears in the automatic and subconscious actions. *Weakened* voluntary or intentional movements are also common motor errors in hysteria. The very fact that the patient's attention is directed to the motor action required, often creates a momentary muscular inefficiency. *Rigidity* and *contractures* may take place in limbs affected by the anesthesia or weakened from the loss of normal motor power. Both the flexors and the extensors are involved and the joint may become fixed in a peculiar way, the same as defined under the accidental hysterical contractures. This state has been termed by Charcot as the "diathesis of contracture."

The *mental stigmata* appear under various degrees of mind perversion. *Forgetfulness* or amnesia is the usual form of disturbance and we may observe this usually as the result of a depreciation in will power or mental concentration. That this loss of memory is purely functional or temporary may be explained by the fact that the perversion is corrected during the subconscious and the hypnotic state. The recovery demonstrates conclusively that the memory impressions are not, in reality, lost. Sometimes these errors of memory are systematized as they relate to a certain class of correlated facts. Again they may pertain only to a certain period of time and for that reason it is termed a *localized* memory loss. In rare instances there may be a *total* or complete loss of memory for a certain length of time, only to be regained by systematic culture and study the same as we require in children. There is sometimes a peculiar *alternation* in memory losses, one group of memory pictures being lost, while another group is regained. *Aboulia* is a term which is applied when the will power is lost and the class of cases suffering with this misfortune lose their determination and power of concentration, and are inclined to be vacillating, impulsive and changeable in disposition and habits. *Suggestibility* is another common mental symptom in hysteria. Under this condition the patient is easily swayed by outside influence or an emotional appeal. There is a tendency to magnify insignificant things; sensitiveness is a marked feature of the temperament and there is usually an absolute dependence upon others. Such patients are rarely able to perform any work. *Simulation* is one of the most common of the mental stigmata, as the hysteria patient is always inclined to mimicry, exaggeration and deceit.

Accidents of Hysteria.—Under this head may be grouped that class of symptoms which refer to the transitory though often severe features of hysteria. They partake mostly of the convulsive character, though motor, sensory and visceral accidents may exist without any evidence of a convulsion. The *hysterical convulsive attack* may take place in a majority of cases without being determined as such on account of the irregularity and diversity of symptoms which characterize it. The *grand attack,* however, which is fortunately a rare experience, presents well defined and extreme symptoms which can not be mistaken. It may be described under four stages: (1) The *prodromal stage,* in which these patients for a time are either depressed or exhilerated. In such conditions they become moody and taciturn or on the contrary they are extremely excitable, irritable and talkative. *Special sense perversions* may now become pronounced and many forms of reflex disturbance may complicate the prodromal state. An *aura* very soon presents itself, either in the form of a wave-like feeling passing over the body, or a sense of constriction or suffocation is experienced and this is often described as a ball rising in the throat, the typical *globus hystericus.* This train of symptoms may continue for an indefinite length of time, with various modifications or exaggerations, until the prodromal disturbance is relieved by the fit, which does not appear with any definite relation to the duration of the previous symptoms. (2) The *epileptoid period* has received its definition from the fact that the symptomatology in this stage has a strong resemblance to the epileptic seizure. The fit is occasionally ushered in by the initial cry, the patient falls, is apparently unconscious and many of the facial signs of epilepsy are present. While the tongue is protruded it is rarely bitten and involuntary urination is uncommon. The tonic spasm of short duration soon ensues, being characterized by slow movements and tetanic rigidity. The feet are extended, the hands are supinated and movements of circumduction are performed such as are never seen in epilepsy. The chest and abdomen are fixed and the whole body is in a state of absolute rigidity. The clonic phase of the convulsion will be defined by the small and quick movements, the arrhythmical breathing, noisy deglutition, etc., all of which actions are independent and illogical. This period usually lasts for six or seven minutes, gradually subsiding into a phase of resolution with stertor, frothing at the mouth and sharp twitchings which show that some of the rigidity remains. A *period of clownism* defines the next phase and is characterized by a series of contortions and wide-ranged or grand movements. *Opisthotonos* or arching of the body upward, the heels and head being firmly fixed on the bed, is one of the extreme contortions of this stage. The body may also undergo marked lateral or forward contortions while alternate flexion and extension of the trunk or limbs, rolling of the head and other grand movements are also characteristic of this stage. The patient during this time seems to be under the influence of some hallucination or delusion, and the violent outcries, the rage, the tearing of garments, the horrible facial grimaces, the biting, scratching and fighting actions remind one of a maniac.

The *period of passional attitudes* follows closely upon the previous period. This seems to blend a feeling of joy and violent passion with the intense state of anger and mental excitement which preceded. In other words the patient seems to be giving a pantomime expression to an apparent intoxication of pleasure or the appreciation of a pleasant dream. This is carried out mostly by postural suggestion, facial expression or the movement of the eyes. This phase of hysteria is, however, not entirely an expression of joy, love and erotism, but has a marked mixture of rage and temporary maniacal excitement.

The *period of delirium* follows soon after the preceding stage and, in a sense, it is a continuation of that dreamy state, though delirium predominates. The patient seems to be dominated by hallucinations and some of the passional attitudes may be repeated. The delirium often takes on the furious form and again it may be characterized by the gay mental state and this at all times fades into a condition of despondency. Many times the actions are similar to those of acute mania, the patient giving expression to obscene and violent language as well as performing many wild and unreasonable acts. This period does not usually last long and the patient soon recovers the conscious state, being somewhat dazed and giving evidence of much fatigue.

The *duration of the attacks* varies decidedly, the entire seizure rarely lasting longer than half an hour and often a shorter time than this. The prodromal and other periods may be prolonged and in exceptionally severe cases one attack may follow another in such rapid succession that a status epilepticus may be simulated. *Modified* or partial attacks may be noticed, in which one or more of the stages may be aggravated, or may alone characterize the seizure are frequently observed. In this respect we may recognize a *vertiginous attack* in which the vertigo is the pronounced feature of the premonitory stage, the vision being somewhat obscured, tinnitus and a sense of cardiac failure also being apparent. The attack stops short of any other stage. In other cases *globus* may be the paramount expression, *tetanic* attacks in which the tonic convulsion is the chief feature are liable to appear, attacks of *passional extreme* characterize some seizures, while *syncope* may be recognized by the extended state of unconsciousness and *somnambulistic* attacks may give rise to some peculiar feats in this line. *Catalepsy* or trance may represent the crisis of an hysterical attack or it may be closely associated with the disease as a matter of habit tendency. Whenever this state exists some evidence of hysteria is usually present to define the cause.

The *motor accidents* of hysteria are represented either by paralyses or contractures, or in some cases by a combination of the two. The paralysis may occur in the form of a monoplegia, paraplegia or hemiplegia following a convulsive attack, a mental shock or traumatism. One marked characteristic is the fact that there is generally an intervening period in which motor disability apparently disappears. Another distinctive feature is the recognition, at times, of some voluntary or automatic motion. *Vasomotor* and *trophic disorders* are usually detected, the electrical and tendon reactions are generally normal, while hyperesthesia and anesthesia, superficial and deep, are distinct characteristics. The intensity of the paralysis will vary according to the hysterical state and the surrounding influences, but usually a permanent recovery is obtained in time. *Hysterical contractures* are in reality caused by the intense rigidity of the muscles, the onset and termination being abrupt, except in rare instances. There is a tendency toward the assumption of certain attitudes, the arm being adducted, with the elbow, wrist and fingers forcibly flexed. Extension seems to prevail in the joints of the lower extremities, while contraction of the spinal muscles may give rise to a pseudoscoliosis. Most of these contractures are transient and the exaggerated ones prevail only during the hysterical attack. Anesthesia, both superficial and deep, is a distinctive feature in most cases and trophic disorders may follow in cases of long standing. There is no marked muscular wasting, the electrical reaction of degeneration is not present and the reflexes are preserved. During sleep and under complete narcosis the contractures usually cease. The hysterical fit, injury or operation are liable to enhance the contraction.

Hysterical hemiplegia is an occasional feature of the disease, though it lacks the cardinal symptoms of the true hemiplegia. The patient seems to jerk or drag the foot in the performance of locomotion, the arm not showing much of the affection. Contracture is often observed in some of the apparently paralyzed muscles. While the *facial muscles* are not much concerned in the hemiplegia they are nevertheless subject to hysterical paralysis or contracture. *Monoplegia* of temporary character may affect any limb, though the muscles of that part are not universally involved. *Paraplegia* is an exceptional complication, and may temporarily paralyze the sphincters.

Hysterical rhythmical spasms of the facial or limb muscles are sometimes recognized and these are, in a sense, a limited feature of the seizure. They may take the form of choreic twitchings, nodding or rotatory spasms of the head, a sharp, barking cough, rhythmical movements of the arms or limbs as if performing a definite function and other movements which are characterized by the rhythmic action. *Hysterical tremors* are of all kinds inasmuch as the patient may counterfeit those which are characteristic of other diseases. We may likewise find them in association with diseases which may be combined with the hysteria or they may occur only during the hysteric attacks.

Sensory Accidents.—These are of transient or periodic character and are usually represented by some form of pain, though vision and hearing may be temporarily impaired. Cephalalgia, generally represented by an area of hyperesthesia, is the most common symptom of this kind. This occurs in periodic attacks and is accompanied by extreme exhaustion, gastric disturbances and other temporary functional imperfections. In case the headache becomes excruciating and there is any sign of delirium, fever or muscular rigidity, it might be taken for meningitis and for this reason this condition is sometimes recognized as a pseudomeningitis. *Spinal irritability* is also a feature of the sensory accidents and the same is true of *visceral neuralgia.* A most typical representation of this form of accident is recognized in the attacks of *hysterical angina,* which may closely simulate the symptoms of a true stenocardia. These attacks are usually induced by some emotion or nervous excitement and precordial hyperesthesia is usually detected in conjunction with the pseudo cardiac neuralgia. The one characteristic differentiation is found in the absence of any real cardiac lesion.

Visceral Accidents.—The peculiar disturbances in hysteria which pertain to the viscera are observed in various forms. The *respiratory* perversions which are not serious are usually recognized in the form of *aphonia* following a temporary laryngeal paralysis. In such instances the patients may speak only in a whisper and yet in an unexpected moment or during sleep the normal voice may return. *Dumbness* is a rare hysteric condition, though this generally is due to a motor amnesia of cerebral origin. *Agraphia* is rarely a feature of hysteria. *Imperfect speech,* however, is frequently induced by the loss of control of the tongue and this is induced by its contraction and faulty protrusion. Thus the patient stammers and becomes extremely nervous in attempting to talk, often giving expression to peculiar noises like a hiccough, sob or grunt. *Hysterical dyspnea* often arises from a sudden contraction of the diaphragm, while in other instances it may appear only as a fit of rapid respirations induced by the extreme nervous excitement. Pulmonary congestion is also a very rare finding in hysteria.

The *digestive accidents* are prominent and numerous, the perversions being manifest in many ways. For instance *anorexia* may be so extreme that the patient often reaches the stage of starvation, and thus emaciation is a frequent symptom. The *esophageal spasm,* which often creates globus, dysphagia. hysterical vomiting and regurgitation of food are characteristic features. The *urinary disturbances* are closely associated with the digestive perversions and partial or complete suppression may attend the attacks of vomiting. *Polyuria* is a marked feature of the prodromes of an hysterical seizure, the patient passing large quantities of pale urine. Evidence of nerve waste is invariably found by the urinary examination and this procedure is frequently a valuable guide in diagnosis as we watch the urinary changes which take place during the paroxysmal stage. Frequent urination is a symptom of the interparoxysmal stage the same as in all nervous individuals.

Hysterical fever may assume various types of exacerbation, and at times it may have a high range. The evidence of infection, however, is not present and, aside from the increase of temperature, the signs of fever are not present.

Trophic and Vasomotor Accidents.—These disorders are not common or enduring. Blood discharges during the hysterical excitement are occasionally seen associated with the perspiration, tears or mammary secretions. Erythematous or vesicular eruptions and neurotic edema are rare manifestations of trophic perversion. Sudden glandular enlargements may precede or attend the seizure or pertain to the interparoxysmal state. This feature is particularly illustrated by the mammary swellings or the hysterical breast which often suggests the possibility of pregnancy. One or both glands may become hard and to a certain extent turgescent and painful, the induration often lasting several weeks or months. Muscular atrophy is also a trophic accident, though it is of rare occurrence.

Prognosis.—The prognosis in the disease must be given conditionally as so much depends upon the nervous makeup of our patient, the character of the past life and the environment. Many of the stigmata may improve under careful treatment, but the disease is liable to continue indefinitely unless the exciting cause is removed. Mild forms invariably respond to internal medication and a change of surroundings. In every case vigorous and persistent treatment, absolute control of the patient and a discipline are necessary to a cure. The disease has a tendency to run a chronic course, many of the sensory stigmata persisting for a long time, while some of the motor accidents are liable to recur at any time. Major hysteria in patients beyond forty is not usually amenable to treatment.

Diagnosis.—In making our diagnosis we must carefully eliminate the nervous diseases and conditions which are easily mistaken for hysteria. The *hysterical temperament* is by no means the disease hysteria and the same, to a certain extent, is true of minor hysteria and its various stigmata. *Hysteria major* presents more serious symptoms and the motor and sensory accidents are of greater importance. This is the real hysteria which demands our consideration and its convulsive features will define the nature of the disease. Inasmuch, however, as mimicry, variability of symptoms and simulation of organic diseases are so natural, a most painstaking differentiation is necessary. We must at the same time remember that serious diseases may be associated with hysteria and we must be careful not to confuse the symptoms of these diseases with those of the latter.

The extreme manifestations of hysteria have their characteristic features. The *paralyses* show no marked wasting, no reaction of degeneration, the deep reflexes are exaggerated or preserved, and other morbid signs of hysteria are always present. The *anesthesia* has its peculiar distribution and the contractures generally cease during sleep, and deep narcosis. The hysterical convulsion should be differentiated from *epilepsy*. In hysteria emotion usually establishes the onset, the movements are coördinate, the tongue is not bitten nor does the patient suffer any injury from the fall. Consciousness is generally preserved and the seizure may be stopped by various methods.

Treatment.—The treatment of hysteria should be undertaken without the aid of drugs. We have made our greatest errors in the management of this disease in attempting to meet a symptomatology rather than to correct a mental condition. In this respect it has been the mistake of the past that we have too frequently relied upon a remedy which seemed to be indicated and overlooked a condition or an influence which has been the creative factor of the disease. Much of this is due to the fact that we too frequently confuse this disease with neurasthenia, which is more amenable to remedies prescribed according to their symptomatic suggestions. The first essential of treatment is to gain the patient's confidence and at the same time to exert a masterful control. The physician who sympathizes and pets his hysterical patients will never attain a result in treatment. The moment the patient has learned the first lesson of obedience to her physician he will be of some help to her after she has determined to get well. Any encouragement of the idea that the patient is an invalid will augment the severity of the symptoms. The fixed idea of infirmity which is the dominant influence must be controlled and corrected.

Sanitarium treatment is not always conducive to a speedy recovery for the comfortable surroundings and the attention of physicians and nurses is liable to magnify the suffering in the mind of the patient. The *rest cure,* which is not made too pleasant and comfortable for the patient, is certainly the correct place for treatment in cases where exhaustion is a feature of the symptomatology. When it is a refuge in which the patient may be petted and encouraged in the nursing of her fancied suffering, it will be of no avail. It is always best to have the patient away from home surroundings where a kindly discipline and a firm influence may help to stimulate a determination to get well and remove the fixed idea of a serious sickness. Physicians are well aware of this requirement, but too few of us seriously carry out this feature of practice, as it is so natural to yield to the influence and the fears of the patient and the family. The very change of locality is often a stimulant which relieves the morbid mentality and when the patient finds no sympathy or is even exposed to ridicule there is often a remnant of self-assertion which is aroused to a proper degree of activity. The comforts of sudden wealth are frequently to be blamed for this affliction. The son or daughter who seeks to purchase pleasure at too rapid a rate simply because they can pay the price are bound to underestimate their physical and mental endurance. In my experience some of the worst forms of hysteria have been observed in the children of wealthy parentage who have been allowed to indulge in social excess, to drink wine and possibly smoke cigarettes, to turn night into day and sleep late in the morning and to encourage every selfish and foolish habit of the so-called ultra-fashionable set. The young man, or rather the young "fellow," who has been allowed the indulgence of a license which presumably belongs to the wealthy class is not only an incurable unless he mends his ways, but is at the same time an object which excites the disgust of any sensible practician. There is at the same time another class of hysterias which arouse our professional sympathy and for whom we should exert our best efforts to save a useful member of society. I refer to that class of noble young women and men whose environment and vocation have demanded an abnormal expenditure of physical and mental energy to gain a living and care for others at the same time. These must in some way be relieved of the intense strain which their occupation and misfortune demand. The reason that the so-called Christian Science has been credited with helping so many cases of hysteria is due to the fact that under this influence the patient's imaginary ills have been supplanted for a faith in which they confidently repose. In some way we must get patients away from themselves and compel them to perform some duty to rid them of this disease. *Hypnotism* has received much praise in the management of these cases, but it is often a dangerous procedure. *Autohypnosis,* or the cultivation of self control, is an important feature of mental culture and should be encouraged in every instance. *Electricity,* massage and all such external measures have only a minor influence in the treatment. Systematic and often vigorous exercise is much better for the patient. This should be indulged to the point of healthy physical exhaustion and so far as possible the exercise schould be a feature of some vocation. When hysterical patients have something to do and are compelled to do it, their work brings them at least temporary mental relief. The majority of these patients become indolent and possessed with a fear of their inability to walk or perform any duty requiring muscular activity. These ideas must be gradually, kindly but determinedly overcome. Hydrotherapy is a valuable tonic and it is best applied as a cold bath or by means of the psychrophore.

The *remedies* which may be used in this disease are referred to under neurasthenia and other subjects. They should be prescribed according to indications after the physician has studied the case long enough to eliminate imaginary complaints. As the disease is in reality a psychoneurosis medication should only be an incident in the treatment. When the patient is made to understand that her physician believes that her condition is not serious enough to demand a prescription, she may overcome her dependence upon medicine. A large sumber of the cases which we see in every day practice have become confirmed hysterics, if not mentally undermined, by strong drugs. The treatment of the seizure may require *nerve sedatives* for temporary relief, but this is mostly for the benefit of relatives. The more we ignore these attacks and the less we prescribe for them the better it will be for our patients, though it is possible that such lack of attention may decrease our popularity.

NEURASTHENIA.

Since this disease was described by Beard in 1869 it has received marked attention by the profession. Under the terms "nervous prostration" and "nervous exhaustion" it has been readily recognized by the profession. It may be a feature of many morbid conditions, but it is probably a neurosis. It is recognized in all of its extremes in the present strenuous business, professional and social life. Nervous irritability and exhaustion are its characteristic features.

Etiology.—Neurasthenia usually occurs after the twentieth year and it is prone to affect both sexes and all classes, though it is most apparent among those whose work and social position demand more than ordinary physical strain. Heredity, occupation, environment, high altitude and debilitating disease play an important part in its development. Mentality is also a factor, as those who have not properly cultivated self-control in work and pleasure are naturally predisposed. Among the *inciting causes* we may mention grief, anxiety, worry and excitement. Overwork and under-rest are frequent factors. Excesses, muscular strain, alcohol, tobacco and many of the foolish habits of the day contribute to its existence. Traumatism, toxemia, anemia and all constitutional diseases favor its development.

Pathology.—While no definite pathologic changes have been discovered it is regarded as a fatigue neurosis. Church says "its manifestations are best explained by a diminished dynamic energy and lessened recuperative power in the cerebrospinal axis and especially in its cellular elements."

Symptoms.—The symptoms of this disease are manifold, and are often exaggerated by the patient. Nerve irritation, exhaustion, headache, backache, gastro-intestinal disturbance, insomnia and depression are most characteristic complaints. The *motor disorder* will be recognized by pronounced muscular weakness, a fine vibratory tremor, facial twitchings and exaggerated tendon reflexes. The *sensory disturbances* are marked by no well defined hyperesthesia or anesthesia. The peculiar *helmet headache* which extends from the occiput over the head, is fairly characteristic. A sense of pressure characterizes this feeling, the intensity being most marked in the back of the head. *Backache* usually attends this and is most marked in the cervical and lumbar regions. Rarely this sensation extends into the limbs, which are usually weakened by exertion. *Tenderness* over the spine is determined by pressure, and is generally exaggerated by the patient. Many peculiar sensations are recited to the physician, the most of which are extremely vague. *Visual disturbances* are common, retinal sensitiveness and perversions of accommodation being sometimes apparent. Photophobia is occasionally present and there may be some impairment of hearing, smell and taste.

The *digestive disorders* are usually marked, nervous indigestion being the most prominent symptom. The appetite is often impaired, constipation is common and intestinal gases will give evidence of impaired digestion. *Cardiac palpitation* is generally detected as a permanent condition or following any unnatural exertion or excitement. The tumultuous action of the heart gives rise to a rapid pulse and many of the features of tachycardia. The pulse is small and rapid, and a cardiac murmur, similar to a hemic murmur, but representing the heart exhaustion, is frequently detected. Pulsating arteries may simulate aneurysm but may be detected in more than one artery. In the majority of cases the feeble circulation may give rise to cold extremities. Because of the feeble circulation and because neurasthenics rarely drink much water, the urine may be scanty. In many instances spasmodic polyuria, particularly at night, is a prominent symptom. *Genital disorders* are characterized by lessened sexual power and spermatorrhea in the male.

The *mental symptoms* are the source of much complaint on the part of the patient though, to the physician, they are of less consequence. Irritability, mental incapacity, loss of memory, a lack of confidence in themselves, depression and a peculiar fear are the prominent symptoms in this respect. Neurasthenics usually have a fear of some impending catastrophe and for this reason they often hesitate to cross the street, to mingle with company or to leave their room. They are always solicitous about themselves. These patients become extremely selfish and their emotional nature is easily aroused. *Insomnia* is always a bothersome symptom. As the result of these mental perversions and the general neurasthenic state, nutrition is greatly impaired.

Diagnosis.—There is no doubt about the diagnosis when the classical symptoms are found. Special significance should be attributed to the fine vibratory tremor, exaggerated knee-reflexes and the occipital headache. *Hysteria,* though often associated with this disease, may be differentiated by its stigmata. *Hypochondriasis* may be recognized by its symptoms of melancholia.

Prognosis.—When the heredity is good and there are no serious complicating diseases a favorable prognosis should be given. Nutritional impairment offers the most discouragement. The vacillating disposition of the patient may thwart the most determined treatment, and an unfavorable environment will always retard the recovery. To accomplish a cure the physician must have absolute control of his patient. The course of the disease tends toward chronicity.

Treatment.—A volume might be written upon the treatment of this disease, but the real object to be attained is *rest.* The *rest cure* is one of our best agencies for this purpose, but this is not within the reach of every patient. It is possible to obtain rest at home under the strict control of the physician, and it will be necessary for the patient to cultivate the habit of relaxation at all times. This may be accomplished when a resolute determination is made not to overtax the strength at any time. It is possible to form a habit in business that will allow a man to work with ease and to confine his labor to certain definite hours. This usually will not be done unless the physician prescribes it and insists upon it as a part of the treatment. When any labor causes physical exhaustion and irritability, it is a sign that shorter hours are required or a vacation is needed. The hurry and worry of modern business and social life is the cause of our large number of neurasthenics. Regular hours for business, meals and sleep are mandatory requirements. Social life, aside from the informal relations of a few friends, must be absolutely forbidden. Diversion is always necessary and, therefore, we must find some innocent pleasure for a regular pastime. Neurasthenia is sometimes due to the lack of occupation and in such cases it will be our duty to prescribe one. In every case the physician must be a good manager or he fails in his treatment. It will never do to sympathize with a patient, for the tendency to despondency is too well fixed. Hope and determination to get well must be inculcated or the cure is retarded. The mental condition is hard to overcome, but this must be done and many times it requires a commanding influence to supplant these vagaries. The patient must become a philosopher and live a simple life to regain perfect health. To impart this influence the physician must possess the same spirit. *Nourishment* is another important essential, as the majority of these patients do not take a sufficient amount of food. A mixed diet with a sufficiency of proteids is always demanded. Digestive disturbances will require tonics and symptomatic treatment to correct these errors and stimulate the appetite. Predisposing diseases must always be corrected or the cause of neurasthenia is not removed. *Massage* and *electricity* probably have their part in the treatment of individual cases, but systematic work or healthful exercise is better for the patient. *Hydrotherapy* is a valuable adjuvant. It may be employed in any way which seems indicated. The cold sponge morning and evening is the simplest form of applying it. The cold bath or the shower will be beneficial to some and when insomnia is marked the psychrophore will be useful.

Powerful drugs are not a part of the medical treatment and usually do more harm than good. *Tonics,* however are sometimes essential. *Iron, arsenic* and *strychnia* may be needed when anemia and exhaustion are pronounced and many of the blood-iron preparations are useful when the hemoglobin is low. The *hypophosphites* are a standard prescription for the nerve exhaustion while *phospho-albumen* and *phosphagon* are also beneficial in this respect. *Strychnia* (grs. 1/60-1/30) may be demanded in case of cardiac weakness, but *strychnia valerianate* (gr. 1/60) is even a better remedy. Iron (gr. 1), phosphorus (gr. 1/100), and arsenic (gr. 1/100) is another serviceable remedy when stimulation is desired. Alcohol should not be allowed. *Sedatives* are often required for sleep and the best of these is *chloralamid* (grs. 15-25) given with a glass of cool water at night. When nervous excitement is extreme the tablet of *zinc phosphid* (gr. 1/8), *cannabin* (gr. 1/10), and *hyoscin hydrobromate* (gr. 1/400) may be prescribed four times daily until the nervous excitement has subsided.

The *homeopathic* remedies are of more importance in the treatment of this disease and we should be guided in their selection by the totality of symptoms. *Zinc* has long been regarded as of first value as it refers to a condition of erethism in the brain and spinal cord. It, therefore, refers to the spasticity, tremors, neuralgia and extreme hyperesthesia. It may be given in the third decimal potency for a long time. *Picric acid* in combination with zinc or iron has a more efficient action when the cell function is extremely impaired or when anemia is a marked factor. The weakness of the limbs, the numbness, the spinal pains, the occipital headache and the profound exhaustion will respond to the use of this remedy. *Ferrum phos., china ars., phos. acid, phosphorus, kali mur., kali phos., china* and similar remedies will have their sphere of usefulness and may be properly compared with picric acid and its combinations. When the innervations are less controllable we shall find use for such remedies as *agaricin, valerianate of ammonia, hyoscyamus, camphor monobromated, gelsemium, belladonna, stramonium, cicuta virosa, enanthe* and *ignatia*. When the mental symptoms are accentuated we shall find no better remedy than *anacardium* which is best used in the second or third decimal potency. The weakness of memory and the periodic signs of irresistible irritation characterize its symptomatology. It has also a marked relation to the digestive disturbances of this disease. Other remedies which may be compared in this respect are *sumbul, asafetida, valerian, moschus* and *coffea*.

When the *cardiac excitement* requires treatment it may be necessary to give it stimulative support by the remedies usually used for this purpose. Generally, however, this is not required as the indicated remedy is sufficient. For the tachycardia there are no better remedies than *fucus vesiculosus* and *lycopus virginicus*.

MIGRAINE.

Migraine has been defined as a constitutional neurosis characterized by periodical attacks of distressing headache. It has also been termed "sick headache," and "hemicrania." Church defines it as "an explosive, paroxysmal psychoneurosis." Aside from the severe headache, nausea and vomiting, mental depression, vasomotor disturbances, vertigo, tinnitus aurium and rarely oculo-motor paralysis characterize the disease. On account of the vomiting it is very frequently regarded as a bilious attack.

Etiology.—Nearly all of the best observers have attributed this disease to hereditary tendencies. It may be related to the grave neuroses or psychoses and it is sometimes closely affiliated with hysteria, epilepsy and rarely insanity. Gout, autotoxemia, injury, shock and neurasthenia are without doubt prominent factors in its etiology. Puberty, adolescence and early adult life furnish the majority of cases and the female is more predisposed than the male.

The *inciting causes* are many. Gastric and intestinal disturbances, eyestrain, grief and emotion are frequent predisposing factors. Lithemia, autointoxication, infections or anything which lowers the physical resistance favors the development of migraine.

Symptoms.—The symptoms of the attack are quite uniform. The *premonitory symptoms* are characterized by irritability, apathy and a dull indescribable feeling which precedes the more severe symptoms. *Sensory symptoms* then develop suddenly. Bright spots, colored rings, luminous zigzags, hemianopsia and other visual disturbances are often marked. The senses of taste and hearing are also affected and other sensory disturbances may characterize the initial stage which is soon succeeded by the headache. *Motor symptoms* may be added to those of sensory character, a paretic condition being occasionally recognized on the side opposite to the hemicrania. Motor aphasia may be present in a few cases while the mental disturbances are rarely extreme in character.

Headache is the most dominant and distressing symptom. It usually starts as a localized pain in the frontal or temporal area and soon involves the lateral half of the head. It may start in the occipital area and extend over the head to one eye or again it may be more diffuse. These attacks last from six to twenty-four hours, and sometimes longer, the patient during this time seeking absolute quiet and freedom from light. The scalp is very tender in the region of the hemicrania and the pain is intensified by any sudden motion and noise. When there is a remission in the headache the patient is extremely exhausted and seeks sleep.

Nausea appears usually as the headache reaches its height and is attended by complete anorexia. Digestion seems to be absolutely interrupted for a time and constipation is generally a feature. *Vomiting* is frequently a symptom and the retching is often extreme. This generally marks the crisis in the attacks.

The *vasomotor symptoms* are marked features of the seizure, a pallor or mottling of the face often preceding the hemicrania. The pallor may be followed by flushing, red streaks often appearing on isolated spots over the seat of the headache. As the attack subsides the surface resumes its accustomed appearance, the pulse becomes normal, diuresis is often marked and as the interparoxysmal stage is approached the patient experiences a marked sense of relief.

The *course* of the disease is characterized by a marked persistency of the paroxysmal return. It seems to resist treatment, but is inclined to disappear as the patient passes middle life or as women get beyond the menopause. A *periodic regularity* is noticed in the return of the paroxysms, though some inciting cause is necessary to start it. The seizures sometimes assume the character of an epileptic attack, or transitory mania and some have regarded migraine as closely allied to the former disease. The *pathology* is not yet understood.

Diagnosis.—The paroxysmal and recurrent character of the headache plus the symptomatology just defined are sufficient to direct attention to this disease. The sensory premonition and the vasomotor features, the hemicrania and the nausea will help to confirm the diagnosis, particularly when we find the hereditary tendency. *Malarial* and *syphilitic headaches* have a more pronounced regularity and the intervals between the attacks are shorter. They have no nausea, vomiting or vasomotor symptoms and there is not a complete recovery after each attack. *Petit mal* may closely simulate migraine, but the aura of epilepsy is much shorter, unconsciousness is always present and headache is not a feature. The headache of *cerebral tumor* is much more severe and depressing, the vomiting is propulsive and without nausea. The headaches of *nephritis* will be differentiated by other symptoms of that disease, the diagnosis being established by urinary analysis.

Prognosis.—Migraine does not readily respond to treatment. Church claims that it "has a tendency to last until involutional changes in the organism commence, when it frequently subsides by a gradual increase of the intervals between the attacks rather than by a lessening of their severity." Removal of the reflex cause and the correction of the neurosis are essential to the cure.

Treatment.—The reflex irritations must first be sought in the treatment. The relief of eyestrain frequently corrects the irritation. Autointoxication is so frequently a cause that we must always take into consideration the possible perversion of intestinal digestion. Occasionally the cause may be found in a gouty or lithemic condition and, therefore, *diet* is an important consideration. The nitrogenous foods must be limited or eliminated on account of this possible cause and it is always advisable to give such patients a sufficiency of pure water and an intestinal antiseptic. *Exercise,* outdoor life, freedom from excitement and worry are essentials in treatment. Constitutional treatment may require such remedies as the *iodids, arsenic, strychnia, iron* and possibly *quinin.* Special remedies like *nitroglycerin* (gr. 1/100), *atropin* (gr. 1/150), and *cannabis indica* (gr. 1/10-1/4) have been used for some time. These may give at least temporary relief in many cases. I have used with some success a compound pill containing *cannabin* (gr. 1/8), *zinc phos.* (gr. 1/10), and *hydrobromate of hyoscin* (1/400). One pill is given three or four times daily until permanent relief is obtained. *Sodium salicylate* or *strontium salicylate* (grs. 10-20) has been successfully used when rheumatism is a factor, and for the same reason *colchicin* (gr. 1/100) has also been prescribed. *Aspirin* (grs. 10) and saline laxatives are equally useful when there is an underlying lithemic condition. The coal tar derivatives may be cautiously used for temporary relief. *Bromid of potash* (grs. 10-20) frequently, and *morphin* (gr. 1/4) occasionally, may be required for the relief of excessive nervousness or severe pain. *Trional* (grs. 15) and *chloralamid* (grs. 15-25) are sometimes demanded for the insomnia which attends the pain. If we give our patient a good night's rest in this way the pain may be better endured while we hold to the remedy which seems indicated. *Caffein* (grs. 2-5) with *benzoate of sodium* (grs. 4) is a popular combination for pain relief. *Guarana* (grs. 10-20) or *menthol* (grs. 5-10), *muriate of ammonia* (grs. 15-30) have been advised by many; these are usually more efficacious when given with hot water. *Local treatments* may include the use of galvanism, the static spark, hot applications, or equal parts of camphor and chloral hydrate may be applied to the scalp.

The homeopathic remedies have been mentioned under neurasthenia and neuritis. *Belladonna, gelsemium, iris versicolor, phytolacca, bryonia, agaricin, zinc phos., cannabis indica, mezereum, physostigma, sanguinaria, glonoin* and *spigelia* have been successfully used when clearly indicated. We must keep it constantly in mind that a careful study of the symptoms will guide us to the use of a remedy which will establish a more effectual relief of the pain.

CHOREA.

The chorea described by Sydenham is the acute form which is generally referred to under this head. It is now classed under the infectious neuroses though by some it is still regarded as an acquired neurosis. It is characterized by irregular jerkings and incoördinate movements. It is a common disease among children.

Etiology.—*Age* is an important factor in the etiology, the largest number of cases occurring before the twentieth year. *Females* are affected oftener than males. It occurs most frequently during the spring and fall. *Inciting causes* frequently include fright, worry, mental shock and overwork. It may be established by traumatism or *reflex irritation.* As examples of the latter cause we may occasionally trace the disease to an eyestrain, an adherent clitoris, phimosis, diseases of the nasopharynx or intestinal irritation. The irritating causes have probably been overestimated in many instances. *Anemia* or *malnutrition* are attending, if not exciting, conditions. The *infectious diseases* of childhood may frequently precede or attend an attack of chorea and this is well illustrated by its intimate relation to rheumatic fever. Just what that relation is has not yet been clearly established, but it is worthy of considerable attention in the study of this disease. *Endocarditis* is also closely allied to chorea, the fatal cases revealing endocardial vegetations similar to those of rheumatic endocarditis. *Pregnancy* is sometimes the cause of severe choreic attacks in women. *Heredity* is always to be regarded as a prominent factor in the development of the disease.

Pathology.—There are apparently no uniform or characteristic changes in the nerve structure. The brain and spinal cord may suffer from intense hyperemia and periarterial exudations. Minute hemorrhages, with secondary softening and emboli, have been found in and about the basal ganglia. Elischer has described "chorea corpuscles" in and around the arterioles and perivascular spaces of the brain and cord. As these are also found at autopsies on septic subjects it tends to confirm the infectious theory of chorea. The cardiac changes include most commonly endocarditis and more rarely pericarditis and mitral disease. Micro-organisms have been noted by several observers, but as yet no specific cause has been decided upon by pathologic investigations.

Symptoms.—While some cases develop suddenly the majority of them are characterized by the *slow onset.* Before the appearance of the motor symptoms it will usually be noticed that the child is *peevish,* there is some *disturbance of sleep,* there is a tendency to *constant restlessness,* and *irritability* is often marked. The child is emotional, capricious or depressed at times. A criticism or reprimand may incite the attack and frequently the premonition of an attack may be noticed in the over-zealous attention to school work or other duties. The child may complain of fatigue, pains in the limbs, loss of appetite and constipation.

The *motor symptoms* of chorea are characterized by irregular and spasmodic muscular movements. These begin in the muscles of the hands and forearm, though the face is often affected first. The child winks, grimaces and drops things from the hands. The *speech* is affected and becomes "halting or explosive" on account of the faulty articulation, due to the involvement of the lips and tongue. The tongue is protruded with difficulty and partakes of the involuntary spasm so that the patient often keeps it in constant motion. *Deglutition* may be interfered with on account of the choreic movements of the lips, cheeks, tongue and palate. The *fingers* show the choreic twitchings early and perform a variety of unnatural movements. The *foot* and *leg* soon are affected similarly to the upper extremities and the child stumbles when attempting to walk. One side is usually affected first and later the opposite side is involved. In about a month the disease reaches its height, the abnormal movements then becoming continuous. The hands can perform no volitional movement, the child must be fed and walking is almost an impossibility. *Respiration* may be involved when the diaphragm and thoracic muscles are affected.

The *choreic movements* may take place both during rest and during volitional acts. In some cases voluntary movements alone incite the incoördinate movement while in other instances the twitchings may occur only when the muscles are at rest. The choreic movements *generally cease during sleep.* The mind is usually disturbed, the patient being irritable, peevish and often suffering with hallucinations.

Pain or *anesthesia* are not usually present. The muscles are weak but not paralyzed. The reflexes may show slight changes. The electrical irritability of the muscles is generally increased, but there are no qualitative changes. Nocturnal enuresis is common and the urine often contains an excess of urea and phosphates.

Cardiac disorders are more common in chorea than has been previously recognized. Functional murmurs are frequently present, inasmuch as anemia and chlorosis are such prominent factors in this disease. Palpitation, precordial pain and arrhythmia may be in evidence according to the severity of the cardiac excitement. Tachycardia is frequently a symptom of prominence. Organic murmurs are always present when endocarditis is a complication. On account of the close relation between chorea and cardiac diseases, the heart should always be kept under close surveillance.

The *general physical condition* in chorea should receive much attention in the management of the disease inasmuch as anemia, anorexia, indigestion, constipation and insomnia so often augment the specific condition. The natural tendency to an associate infection will also give rise to many unexpected symptoms, a sudden fever occasionally appearing.

The *course* of this disease is characterized by exacerbations and remissions. The average duration is from four to eight months, though aggravated cases may last much longer. Improvement usually appears gradually, the lower extremities being relieved first. The upper extremities, the trunk, face and tongue then improve in the order named. A recurrence is liable to be experienced from the slightest perversion of health or nerve exhaustion.

Other forms of chorea have been described. The *grave form,* or chorea gravis, is characterized by an aggravation of the motor symptoms and in many instances of the mental symptoms as well. It may appear as a recurrence of the common type in aggravated form. The *onset* is usually sudden, the choreic movements are on a large scale, continuous and violent. Locomotion is often impossible, deglutition is performed with difficulty and speech is incoördinate. The patient is liable to injury by a sudden fall, fever often appears, delirium is not uncommon and the patient may become maniacal, the choreic status ensuing.

The *chorea of pregnancy* is noticed in young primiparae, but generally reveals a previous history of chorea. In addition to the characteristic symptoms of the ordinary form of the disease, mental disturbances are pronounced. The choreic symptoms rarely disappear before parturition and even then they may continue. They may recur at later pregnancies and a fatal termination is not uncommon.

Paralytic chorea has also been termed "limp chorea" inasmuch as the paretic symptoms are more pronounced than the choreic movements. Young children—four or five years of age—are most frequently predisposed, an infection being frequently the inciting cause. Monoparesis is most common, though hemiplegia or paraplegia are of rare occurrence.

Diagnosis.—There is rarely any doubt about the diagnosis in the ordinary type of chorea, as the symptoms are so clearly defined. The choreic movements which attend other diseases are often confusing. Athetosis, posthemiplegic chorea, Friedreich's ataxia and the rhythmic tremors of hysteria have been differentiated in their appropriate place. The *tics* appear more suddenly and are recognized by their subconscious character. *Huntingdon's chorea* has a family history, appears later in life and shows a tendency toward dementia. *Myoclonus* and the *electric chorea* of Dubini have but slight similarity to chorea.

Prognosis.—This is one of the diseases of the nervous system which will respond to early treatment. In a sense it is a self-limited disease. Recovery depends largely upon the previous history and the general health of the child at puberty. The intensity of the attack, the violence of the choreic movements and the evidence of mental impairment are always unfavorable symptoms. Cardiac complications, infection, pregnancy and the presence of the choreic status should give an unfavorable prognosis.

Treatment.—The treatment is largely a matter of *rest.* The muscular weakness renders this necessary while the mental enfeeblement also calls for relaxation and diversion. The child should be taken from school, duties which become onerous and exhausting should be temporarily suspended and the hours for rest should be prolonged. The environment must be such that excitement is not present and nervous relatives should not associate with the choreic child. *Enforced rest* is often required and for that purpose it has been my custom to give many of my extreme cases the wet pack during the middle of the day, the sheet being wrapped about the body with such firmness that jerking is absolutely impossible. Usually in such treatments the child sleeps quietly for an hour or two and then is not exhausted and irritable before the regular time for retiring. *Insomnia* is many times a troublesome feature, though usually the child sleeps from sheer exhaustion, the twitchings ceasing at this time. In case sleep does not come naturally it is proper to give a mild sedative until the rest at night is normal. A small dose of *chloral hydrate* (grs. 5-15) may be given during the early evening and repeated later if necessary. *Hyoscin hydrobromate* (gr. 1/500-1/200) is also a valuable sedative which quiets the nervous system and after a time permits a natural sleep. If one dose in the evening does not obtain the desired effect, it should be given two or three times daily. *Chloralamid* (grs. 5-15), *trional* (grs. 5-10), or perhaps smaller doses, will often insure sleep at night, and the natural sleep may then come on succeeding nights. It must not be understood that hypnotics are the best form of treatment, but we must give the patient sleep or else our remedy will not always accomplish what we desire and expect.

The *diet* is another factor of importance in the treatment of chorea. Nutrition should be maintained by easily assimilated food inasmuch as digestive disturbance is common in this disease and is sometimes the inciting cause. Fats should be a prominent feature of the dietary for the sake of trophic improvement.

Specific treatment will include first of all the use of *arsenic* for the sake of the anemia and nerve depletion. With this it is often wise to use iron. For that reason a pill containing *iron* (gr. 1), *arsenic* (gr. 1/100), is a very good combination to be given three or four times daily. *Phosphorus* (gr. 1/100) may be combined with this or it may be given alone with equal frequency. *Fowler's solution* of arsenic is still the popular remedy with many practicians and I am convinced that in many instances good results are obtained by its use. It is generally given in three to five drop doses after each meal. Gastric disturbance will contraindicate its use. Remedies like antipyrin or phenacetin (grs. 5), antifebrin (grs. 3); exalgin (grs. 3) have been advised by some, but their temporary relief of pain is the only excuse for their use, and the cardiac depression which often results renders them undesirable. *Cimicifuga* (grs. 1-2) three or four times daily is an excellent remedy in young girls who are approaching puberty. *Zinc valerianate* (grs. 1-2) three times daily is another valuable remedy when there is an underlying or inherited neurasthenia. The *bromid* of *zinc* (grs. 1-2) is also regarded favorably by some. Small doses of bromid of sodium or potassium are also standard remedies. The following combination which has rarely failed me in the treatment of this disease is *zinc phosphide* (gr. 1/10), *cannabin* (gr. 1/10) and *hyoscin hydrobromate* (gr. 1/400). One of these pills is given every three or four hours in severe cases and gradually decreased in frequency as improvement is apparent. Patients of highly nervous temperament who are greatly exhausted will respond to the *valerianate of strychnia* (gr. 1/100-1/60) three or four times daily.

Local treatments often give assistance in many cases. The girl with a hooded clitoris, or the boy with a phimosis certainly need attention in this respect, but we should not place too much stress on this relief of local irritation. For the same reason a young woman may need uterine treatments and where it is absolutely necessary such procedure should not be neglected. The tendency, however, in this disease, is to place too much stress on these features. There is no doubt that some relief is obtained by stretching the rectal sphincter, but uncalled for claims are too frequently made for such adjuvant treatments. The same is true regarding eyestrain; many choreic patients are helped by the relief obtained from glasses, but we must remember that this reflex irritation does not alone cause chorea and the correction of eyestrain does not alone cure the disease.

Hydrotherapy offers much in the treatment of this disease. The use of the *psychrophore* is one of the best adjuvants. Ice water should be allowed to slowly run through this after it is inserted into the rectum, the patient lying on the left side and controlling the flow of water by holding the tube in the hand. The *cold sponge* and the cold pack, as previously described, are of much service in relieving the nervous system of its constant strain. The *hot bath* is often of more value by its sedative action. This may be given at night if the patient is inclined to insomnia.

Physical exercise is of value so far as it relieves nerve strain. If this may be taken as play the mental relief will be as helpful as the exercise itself. It is an error, however, to permit violent exercise or excesses of any kind. *Mechanical therapeutics* may often be advantageously employed. *Electricity,* in the form of a static breeze, has been helpful in many cases. *Bad habits* in children must always be looked for; the correction of these may be of greater help than the use of remedies. Overeating, eating between meals, late hours for going to bed, evil associates, cigarette smoking and masturbation are only a part of these errors which a careful physician must overcome.

The administration of homeopathic remedies has been very satisfactory in the treatment of this disease. With these and the help of the adjuvant suggestions above I have usually succeeded in curing the majority of my cases. *Agaricin,* an active principle of agaricus muscarius, has been a most valuable remedy in my practice. I invariably give this in the third decimal potency, four to six times daily, and it has cured most of my cases. This remedy in its proving not only presents the characteristic symptoms of this disease, but spinal anemia often results from its prolonged use. As this is, to my mind, one of the marked pathologic conditions in this disease, it furnishes an additional reason for the administration of agaricin. With this remedy we should always compare *camphor, coffea, pulsatilla* and *cannabis indica.* The next remedy of importance in my treatment is *valerianate of ammonia,* which is so commonly used in cases of neurasthenia and nervous irritability when other remedies fail. I give this in the second decimal potency several times daily as the case may need. *Asafetida, ignatia* and *moschus* may be compared with this. *Tarantula cubensis* probably ranks next in its efficiency, but we are not always sure of getting a reliable preparation. When this is indicated the chorea is more of the hysterical character, while dysmenorrhea and spinal irritability are also marked features. The sixth to thirtieth potencies are probably best when this is used. *Cimicifuga* is a good remedy for girls inasmuch as it has a powerful action upon the cerebrospinal and muscular system as well as upon the uterus. I prefer this in the first or second potency. *Caulophyllum, pulsatilla, aletris* and *gelsemium* may be compared. *Hyoscyamus, belladonna* and *stramonium* will be indicated in a variety of cases according to symptoms previously outlined in comparison of these remedies. *Ferrum picricum, ferrum phos., phosphorus, phos. acid, kali phos.,* and *zinc phos.* will be required in many cases when chlorosis and anemia are factors. *Coffea* is frequently indicated in those cases when there is extreme sensitiveness or when insomnia prevails. *Cina, cypripedium* and *chamomilla* will correct many cases when intestinal disturbances and worms are the source of irritation. *Conium* is of occasional service when there is pronounced physical exhaustion with mental depression. *Anacardium* will be thought of when the mental state is excitable. *Crocus sativa* is often useful when the spasmodic contractions seem to attack a single group of muscles; hemorrhage is often a factor in the symptomatology of this remedy. *Cicuta virosa* will often be indicated when convulsions attend the choreic symptoms and when the mental phase assumes the character of melancholia. *Strychnia, enanthe, kali bromatum, belladonna, hyoscyamus, stramonium* and *agaricin* may be compared. *Mygale* is often useful in mild cases of chorea when there are only slight twitchings of the facial muscles. *Cuprum* has a similar usefulness, but muscular cramps are liable to attend. *Thaspium aureum* may be compared, but this remedy usually refers to choreic manifestations which occur during the evening or in the night. *Sumbul* has a sphere of usefulness which relates to nervous symptoms which resemble those of hysteria with sensations like globus, nervous palpitation of the heart and mental excitement.

HUNTINGDON'S CHOREA.

This is a chronic progressive chorea which was defined by Huntingdon as an hereditary disease. It has also been described under such terms as "chorea of the aged," "family chorea" and "adult hereditary chorea." It is characterized by irregular muscular contractions, incoördination with a tendency to mental deterioration, and dementia.

Etiology.—The disease is known only as an hereditary affection, being often traced back for four or five generations. It seems never to reappear after the hereditary chain has been broken. It is supposed to alternate with idiocy, epilepsy and degenerative conditions. In certain localities it has appeared in endemic form. The male sex is most frequently affected and it is very rarely seen before the thirtieth or fortieth year.

Pathology.—No characteristic pathologic changes have been observed except those which are common to dementia. Pachymeningitis, leptomeningitis, cerebral atrophy and compensatory hydrocephalus are mentioned by Church. A mild degree of sclerosis has been observed in the pyramidal tract, the anterolateral and the cerebellar tracts occasionally partaking of this change. Neuroglia proliferation may take place in the cortex and a more general cellular degeneration of the cortex cells is possible. This is most prominent in the frontal area.

Symptoms.—These appear insidiously, a mental enfeeblement often preceding. The facial muscles manifest a slow involuntary contraction, the speech is thickened and drawling, a hand or a finger may suddenly contract and locomotion is often performed only with deliberation. The gait becomes more erratic until that of a drunkard is simulated. Emotion and embarrassment may aggravate these abnormal actions until the patient assumes peculiar gestures, poses and exaggerated muscular actions. The *mental symptoms* resemble those of dementia, irritability, maniacal violence and delusions, often revealing the perversion of intelligence. The *course* of the disease tends toward chronicity though the patient may live to a good age.

The *diagnosis* is not confusing when the family history is obtained. *Chorea proper* will present a different history. the mental phase being absent while its acute manifestations are more marked and recovery is possible. The *family ataxias* show no mental degeneracy and appear earlier in life. *Tics* have no symptoms of dementia, the rhythm of the movements is more active and the movements are limited. *Rhythmic chorea* appears in conjunction with hysteria, single muscles or groups of muscles being affected. The *treatment* must be symptomatic, but improvement is not to be expected.

CHOREIFORM AFFECTIONS.

Myoclonia.—Unsystematized, involuntary, arrhythmic and quick muscular contractions have been defined under the terms myoclonus or paramyoclonus. These sudden contractions may affect a muscle group, a single muscle or only a few muscular fibers in a given muscle. Under myoclonia are included *paramyoclonus multiplex,* the *electric chorea* of Bergeron and Henoch and the *fibrillary chorea* of Morvan. Epilepsy is sometimes combined with myoclonia.

The *etiology* is not understood. Lundborg claims it is related to thyroid disease, and a nervous heredity is supposed to predispose. Overwork, traumatism, fatigue, emotion and other factors have been considered. The *pathology* is also unknown, though the cortex motor cells are supposed to be disturbed.

Symptoms.—The *motor symptoms* may follow an insidious development or a sudden onset. The *contractions* which are clonic in character, usually begin in the lower extremities, are bilateral though not always symmetrical, and soon involve the upper extremities; the face is rarely affected. These contractions are usually involuntary, though they may be somewhat controlled. Use of the involved muscles may cause a temporary cessation of the abnormal contractions. The clonic spasm may in time assume tonic or tetanic characteristics. In contrast to the irregular and arrhythmic contractions, a constant agitation may be noticed in rare instances. The contractions usually cease during sleep, though this is not always the case. A sudden emotion or a physical disturbance may excite the muscular action. The *mind* is not generally affected, the face, tongue and trunk also being usually spared.

The differential diagnosis must consider *chorea electrica* and *tics*. The *prognosis* is fairly good. *Treatment* should be symptomatic.

Dubini's Disease.—This has been erroneously termed *chorea electrica*. The disease is not properly allied to chorea and has also been compared with myoclonus and hysteria. The *etiology* and *pathology* are not fully understood, though infections, particularly malaria and typhus, have been given as causes. The conditions which are incident to pulmonary infections, splenic congestion, meningitis, basilar cerebral congestion and cortex softening have been mentioned as pathologic findings.

The *symptoms* are characterized by an abrupt onset, pains beings felt in the head, neck and lumbar muscles. The extremities are soon affected by short and sharp spasms which give rise to the term "electric chorea." These symptoms generally start in the hands, fingers and face, pain being marked in those parts. Later the symptoms may assume hemiplegic or diplegic characteristics. Epileptoid convulsions sometimes appear, though there is no loss of consciousness. Regular intervals usually occur between the spasms.

The *course* of the disease tends toward a progressive aggravation of the symptoms. The spasm may become so severe that death occurs during some of the extreme convulsions.

Chorea Associated with Hemiplegia.—Chorea affecting one side of the body may anticipate an attack of hemiplegia. The choreic movements usually begin a few days before the apoplexy, but disappear with the appearance of the paralysis. *Post hemiplegic chorea* appears on the hemiplegic side soon after the apoplectic attack. This is usually permanent. *Athetosis* is closely allied to post hemiplegic chorea and may be considered as a characteristic symptom of this condition. It is defined as "a slow, successive flexion, extension, pronation and supination of the fingers, hand and arm or of analagous movement of the toes and feet." The motion rarely ceases during the waking hours. The contractions are forcible, steady and sometimes painful. The lesion is said to be in the optic thalamus, though it often follows cerebral infantile palsy. The *prognosis* is usually unfavorable as regards cure. The *treatment* pertains to the causative disease.

Habit Spasm.—This is not a true form of chorea, though it is characterized by localized contractile movements. The neuropathic state usually precedes this and it is liable to occur in early life as the result of overstudy and nerve exhaustion. There is usually an attending deterioration of the general health.

The *symptoms* are characterized by spasmodic movements of certain muscles. Grimaces, excessive winking, twitching of the mouth, shrugging of the shoulder or jerking of the head may be observed, though usually only one of these symptoms is recognized.

The *prognosis* is usually favorable if the underlying cause can be found and removed. The treatment must pertain to the general health, the environment and the acquired habit. General and symptomatic treatment will be similar to that of chorea and neurasthenia.

General Tic.—While this disease closely resembles habit spasm and mimic spasm it is considered as a psychoneurosis which is characterized by coördinate spasmodic movements. These spasms may appear in the form of an uncontrollable winking, sudden movement about the mouth or nose, swallowing efforts or the production of abnormal sounds. The neck and upper extremities may be involved so that attitudes and gestures are developed. Some underlying mental idea seems to provoke these spasms. These tics are *rapidly executed* and are usually frequently repeated. Excitement or embarrassment often develops them. They are generally clonic in character, though tonic features may appear. The spasm may affect neighboring and associate muscles and rigidity may be observed when the neck muscles are involved. The young are usually affected more than adults, though the latter are not always exempt.

Several varieties may exist and among these may be included the degenerate tics, blepharospasm, facial spasm and spasmodic torticollis, some of which for other reasons have been included under special headings. A form known as *mental torticollis,* characterized by a deviation of the head which is spasmodically maintained during excitement, has been mentioned. This usually ceases from pressure and lying down. A *saltatoric spasm* or "jumpers" is a peculiar clonic contraction occurring in the lower limbs. Upon command these patients may strike, jump or even unclothe themselves. The *prognosis* in these cases is usually favorable, the disease usually responding to symptomatic treatment.

PARALYSIS AGITANS.

Paralysis agitans or Parkinson's disease has been commonly termed "shaking palsy." As the tremor often appears late in the disease it is frequently confused with the symptoms of senility. It is considered usually as a neurosis, being characterized by a typical tremor, a peculiarity of speech and gait, with a loss of muscular power as the disease progresses.

Etiology.—The disease is one of *late middle life,* rarely appearing before the fortieth or after the sixtieth year. *Males* are affected most frequently and a neuropathic inheritance is commonly found. *Exciting causes* include physical exhaustion, fright, anxiety and grief. Other etiologic factors are not apparent.

Pathology.—Lesions, aside from those of senility, are unknown. "Changes have been observed in the encephalon, cord and muscles." Scleroses have been found in the motor cells of the anterior horns and in the fiber tracts of the cord. Dana has described "a diffuse sclerotic myelitis with vascular lesions and cellular atrophy in both the cord and the cortex."

Symptoms.—*Muscular rigidity* is one of the early symptoms. This causes slowness of movement, peculiar attitudes and postures and the immobile face. It may appear with or without tremor, but is generally detected first in the parts which present tremor. The tendon reflexes are only slightly increased and foot clonus is not present. The *facies* is characteristic. The lines of expression are removed, the eyes are wide open and the patient rarely winks. For a more perfect vision the patient "turns bodily with the neck held rigid." The *speech* is monotonous and deliberate and soon assumes the scanning character. The *neck rigidity* simulates a condition of torticollis. The *body* is bent forward and the arms are flexed at the elbows and removed from the sides, the fingers being held in tight flexion. The lower extremities do not, at first, manifest the same tendency to flexion, though this may appear later.

The *gait* of paralysis agitans is peculiar. Hesitation marks the first effort to walk and when the locomotion is well induced a "trotting gait" is noticed as the patient aims to overcome the body flexion and the muscular rigidity. The propulsive character of the gait is observed when locomotion is well under way and it is often necessary to stop the patient by force.

The *tremor* appears with the rigidity or after it has developed. It usually begins in *one hand* and *arm* and then gradually extends to the same parts of the other side, the limbs being affected rarely and late in the disease. In the hands it is characterized by a "rhythmical, alternating flexion and extension of the fingers" which soon gives the "pill rolling" motion. Flexion and extension of the wrists with movements of pronation and supination may be added. In the *lower extremity* the tremor, if present, will be noticed in the ankle, the foot constantly tapping the floor. The thigh muscles may rarely show the tremor. The *head* commonly shakes with the tremor of the other parts, though there may be a "rhythmic nodding, shaking or rotation." The tremor may continue during rest of the involved muscles or when the parts are supported. It rarely simulates the intentional tremor of multiple sclerosis. Sleep usually causes the tremor to subside. The tremor is distinctly shown in the handwriting. *Sensory disturbances* are not pronounced and the palsy rarely attains the degree of complete paresis. Extreme weakness, however, is common in advanced stages. Muscular atrophy is not a feature of the disease, electrical changes are absent and sphincter weakness is not a factor. The mental condition may simulate dementia, but this is not usual.

Diagnosis.—A mistake in diagnosis is rarely made if the characteristic symptoms are observed. *Post-hemiplegic tremor* may resemble it, but this is unilateral and has a history of apoplexy. *Senile tremor* does not have the same rigidity, the head is usually affected most, but the facies is absent. *Multiple sclerosis* has an intention tremor and the reflexes are exaggerated. *Hysteria,* with its stigmata, presents so many different symptoms that it should rarely be mistaken for paralysis agitans.

Prognosis.—So far as improvement is concerned the prognosis is unfavorable as the disease may have a prolonged duration. The patient may become helpless in advanced age.

Treatment.—This is purely symptomatic as the disease can not be cured by remedies or local treatment.

TETANY.

Tetany is characterized by tonic contractions which commence in, and are usually confined to, the muscles of the extremities. It is associated with sensory and motor disturbances with malnutrition as an underlying disorder.

Etiology.—On account of its occasional epidemic occurrence it has been regarded as an infectious disease. Some have regarded its epidemic appearance as an expression of hysteria rather than of tetany. There is more positive evidence of its infectious character, as it is often associated with infectious diseases. Gastro-intestinal intoxications are without doubt frequent causes, hyperacidity, fermentation, diarrhea and chronic constipation often being the symptoms which appear in conjunction with the tonic cramps. Pregnancy and lactation may be predisposing causes, and there is evidently a close relation between this disease and myxedema. Tetany has followed enucleation of the thyroid gland. The disease is not common in this country except in the crowded centers where the foreign population is dense. Sarbo claims that impaired nutrition is the most common cause. It is practically a disease of middle life, as few cases are observed after the fiftieth year. Both sexes are alike affected. *Morbid lesions* have not been found, though slight vascular changes in the brain and cord have been noticed.

Symptoms.—A *spasmodic stiffness* is first observed in the fingers or toes, though it may affect the parts below the elbow and knees. Usually the knees alone are affected, yet the spasm may gradually advance toward the trunk, while in other instances it is confined to the upper extremities alone. The *onset* may be characterized by headache, malaise, pains, paresthesia and premonitions of the approaching rigidity. *Pain* becomes a factor as the spasm gradually increases in intensity, though there may be temporary intermissions in the rigidity.

The *attitudes* assumed by the parts undergoing spasm are quite characteristic. The hand has been likened to that of the accoucheur, the fingers being straightened, bunched and flexed upon the hand. The hand may be drawn to the ulnar side and the wrist is often flexed. The feet assume the position of equino-varus with flexed and overlapping toes. The elbows are flexed and drawn close to the sides. The knees and thighs are rarely flexed except in extreme cases. The muscles undergoing spasm are very tense, firm and often the seat of pain; no voluntary motion in these parts is possible and pain follows from any forced movement. Opisthotonos may occur and dyspnea is a prominent symptom when the spasm invades the trunk muscles. The eye and face muscles may become involved, while the larynx and esophagus may be similarly affected. The sphincters are also liable to suffer from the spasm. Fibrillary twitching may be occasionally observed in the contracted muscles.

The *symptoms of the interval* are characterized by peculiar phases of mechanical and electrical irritability. *Trousseau's sign* is the causing of an attack by pressure upon the nerve or vessels of the limbs. *Chvostek's sign* is the causing of a facial contortion when the trunk of the facial nerve is excited by a slight hammer stroke. *Erb's sign* is recognized as the increased electric excitability of the nerve; this is particularly true of the galvanic current. *Pain* is a common symptom when there is any pressure upon the involved nerve and it also "induces a persistent paresthesia in the cutaneous distribution." *Edema* is often noticed in the hands and feet and the face may be slightly affected, giving rise to an expressionless appearance. The *sensory disturbances* are often prominent, a slight diminution in sensibility being observed in the affected limbs. Hemianesthesia and symmetric areas of insensibility to pain and touch are occasionally present. *Trophic changes* are also in evidence, herpetic eruptions often appearing along the course of the involved nerve. *Fever* is quite common. The *urine* usually shows a large amount of phosphates, and indican is sometimes present.

Diagnosis.—The character of the spasm and the hyperexcitability of the muscles and nerves should settle the diagnosis. Trousseau's symptom is a valuable diagnostic sign. The facial phenomenon is also a distinctive symptom. From *tetanus* it is differentiated by the less pronounced spasm, the intermittency of the contractions, by the appearance of the spasm in the extremities and the absence of trismus until late. None of the distinctive phenomena of this disease are seen in tetanus. *Meningitis* has none of the special signs of tetany though when it occurs in infants it may be mistaken for this. *Hysteria* should be differentiated by its stigmata.

Prognosis.—The prognosis is usually favorable if the underlying cause, malnutrition, may be overcome. The majority of cases pursue a mild course. Pregnancy and lactation also unfavorably predispose. When the tetany occurs during labor a fatal result is not uncommon. Serious attending symptoms like respiratory excitement with the extreme spasms, cerebral symptoms and uremia are always to be feared.

Treatment.—The spasms may require sedatives or morphin. *Hydrobromate of hyoscin* (gr. 1/400), *zinc phosphide* (gr. 1/10), *cannabis indica* (gr. 1/8), one pill every three or four hours, is a valuable combination for consideration in this disease. *Bromids* and *chloral,* in moderate doses, may often be sufficient to quiet an attack. Massage, electricity, hot and cold baths may also be considered in the treatment. Attention should be given to the cause more than the condition. Otherwise the treatment is purely symptomatic.

FATIGUE NEUROSES.

The overuse of certain muscles which is demanded by various occupations may create what is termed a fatigue neurosis.

These are often described so as to suggest the vocation or the muscular movements which are performed. They are characterized by pain, tremor, cramp and muscular weakness.

Writer's Cramp.—Etiology.—This is the most common form of fatigue neuroses. It appears in that class of people whose vocation requires the constant use of the pen or pencil. In the present age we may observe a similar condition following the incessant use of the typewriter. A *neuropathic* history usually precedes and heredity may be an occasional factor. *Sex* is not a factor, though we observe most cases among girls nowadays from the use of the typewriter. *Injury of the hands* may be a rare cause.

Pathology.—While many theories prevail regarding the pathology it is quite certain that we must exclude the muscles and nerves of the part involved. The changes, as claimed by Hodge, are probably located in the motor cells as a result of the persistent physiologic action. As muscular atrophy occasionally follows the spasm of long standing we should naturally locate the lesion in the motor cells of the cord. The sensory symptoms disturb this theory somewhat.

Symptoms.—The *onset* is rarely noticed as it appears so insidiously. The patient will often first complain of *numbness* and weakness in the fingers of the hand which is overused; a *tremor* in these parts gradually ensues unless the occupation is changed. The *impairment of function* is characterized by the spastic condition of the muscles involved and the tremor. A neuralgia may appear in some cases and the disease may progress to complete palsy of the affected parts.

In *mild cases* the spasm only appears during the act of writing, but in severe cases it may persist without any local excitement. The muscular weakness is followed by atrophy of the muscles in some cases. In such instances the reaction of degeneration will be noticed in the muscles of the affected hand and fibrillary twitchings of these parts may appear. The *sensory disturbances* are characterized by fatigue, pain or tenderness. These symptoms are excited when the hand is used and in severe cases may continue after. The abnormal sensations soon extend to the arm.

Diagnosis.—This disease is often mistaken for arthritic disturbances, musculo-spiral palsy and neuralgia. Monoplegia or hemiplegia from organic causes may sometimes be mistaken for this disease.

Prognosis.—This is unfavorable, inasmuch as cases rarely reach the physician before it is too late for a cure. It is otherwise unfavorable because many patients can not well change their vocation. The neuralgic phase is more responsive to treatment than the condition of spasm.

Treatment.—The occupation must be changed or a splint must be used on the hand in order to call the arm muscles into use. Massage, electricity, physical exercises and symptomatic treatment may be of some help.

FAMILY PERIODIC PARALYSIS.

This, according to Church, is characterized by recurring attacks of flaccid motor palsy with diminished reflexes and decreased electrical excitability. The palsy affects chiefly the lower extremities.

Etiology.—The prominent factor is heredity. Both sexes are affected alike and the disease usually appears in the early part of life, children under ten years being most frequently afflicted. Fatigue and mental excess are the common exciting causes. Goldflam claimed an autotoxic cause.

Symptoms.—The attacks occur most frequently at night, developing slowly. Paresthesiae occasionally precede the attack, headache, backache, coldness of the limbs and rapid heart action sometimes being observed. The palsy affects the lower extremities chiefly, but may involve all of the muscles of the body except those of the head. The attacks may occur frequently or at rare intervals, the paralysis disappearing in the reverse order of development. The tendon reflexes are lessened or lost during the attack. The electrical reactions are also lost.

The *diagnosis* is made by the family history, the periodic recurrence of the palsy, the loss of the reflexes and electrical reaction, and the absence of sensory and mental disturbances. A *family tremor* which affects the hands, the head, face and tongue must be differentiated. The *prognosis* is not unfavorable to life. The *treatment* must consider the cause and the heredity, though remedies rarely apply.

THOMSEN'S DISEASE.

Thomsen's disease, or myotonia, has received its name from the physician who, suffering from this disease, described this particular neurosis. It is a rare disease, which is manifested by a peculiar rigidity of the voluntary muscles when motion of these is undertaken after a period of repose.

Etiology.—This is an hereditary infirmity, most of the cases appearing in family groups. Males seem to be most commonly predisposed.

Pathology.—The pathology is closely related to a myopathy, though it has been regarded as a trophoneurosis or a "congenital defect in the trophic and motor apparatus of the cord." (Church.) The muscle fibers are increased in their transverse diameter, there is an increase in the number of nuclei, and sometimes the muscle fibers are vacuolated. A slight increase of connective tissue is noticed.

Symptoms.—There is a prolonged contraction of the muscles when motor action is undertaken after repose. In attempted motion an apparent rigidity interferes; after a time this disappears, though the spasm may be repeated by unnatural and sudden movements. All the voluntary muscles are liable to be involved; the sphincters and unstriped muscles, however, are not involved. The affected muscles give evidence of pseudo-hypertrophy with diminished motor function. While the reflexes are normal a spasm may follow from a slight excitation of the tendon. *Psychic disorders* may attend these symptoms. The *diagnosis* must differentiate tetany and pseudo-hypertrophic paralysis. The *prognosis* is unfavorable. *Treatment* must take into consideration the causes of the myotonic cramps and aside from massage and electricity, is purely symptomatic.

EXOPHTHALMIC GOITER.

Exophthalmic goiter is often included under the diseases of the thyroid gland, but it is properly a trophoneurosis. The same is true of myxedema, but on account of the thyroid changes this is kept under the classification of ductless gland diseases. Exophthalmic goiter is also known as Graves' disease and Basedow's disease. It is recognized by its classical symptoms, tachycardia, a fine tremor, enlarged thyroid and exophthalmos, though either of the last two symptoms may be absent in some cases. Added to this we shall usually observe many symptoms in common with neurasthenia.

Etiology.—The disease occurs with most frequency between puberty and the menopause, and women are usually affected. It is rarely observed in men. A neuropathic inheritance is common, neurasthenia being a frequent precursor. Debility, chlorosis, menstrual irregularities and pregnancy are closely allied. It may start from a nervous shock or pronounced grief. It may be rarely related to epilepsy, chorea and hysteria. A family type is sometimes seen. It has always been held that it has a close affinity to changes in the thyroid gland, but this has not been satisfactorily established.

Pathology.—The pathologic findings are not constant. The *heart* may be dilated or hypertrophied, though often it reveals no pronounced changes. The *thyroid* gland usually shows a goitrous enlargement, though this may be wanting. Its blood vessels in chronic cases may be thickened, atheromatous and dilated. The acini of the glands may also be dilated, the colloid material may disappear and the secretion is often altered. The *thymus gland* may be enlarged. The orbital cavity frequently presents an adventitious increase. The *nervous system,* which is probably most concerned in the disease, does not present the changes which would be expected. The theory of sympathetic irritation and alterations in the cervical sympathetic ganglia, which is most probable, is not substantiated by pathologic investigations. Brain and cord changes are also wanting. The associate lesions of tabes are claimed, but no symptoms seem to confirm this. Atrophy of the restiform bodies has also been claimed. In fact no specific pathologic features which are peculiar to this disease have been found.

Symptoms.—The symptoms usually develop insidiously. The disease is often not recognized by the inexperienced observer until the cardinal signs are present. The *tachycardia* is generally the first disturbance which sends the patient to the physician. The pulse is regular but rapid, and may be as high as 120 per minute in the early stages and 140 to 160 in the later stages of the disease. Cases are reported with a pulse rate as high as 180 to 200 per minute. The tachycardia, aside from the general nervousness, may be the only special symptom throughout the disease. The tumultuous action of the heart may lead to arrhythmia, while precordial distress, vertigo and sometimes cyanosis may attend. Palpitation is usually experienced on slight exertion and functional murmurs may appear in severe cases. Organic disturbance may complicate the tachycardia, but this is not caused by this disease. Capillary pulsations are often pronounced and they may be observed over the liver and spleen.

The *goiter* is usually insidious in its development or it may not be present. Rarely it is of sudden development. It is exceptional for the goiter to assume a large size. A pulsation is usually felt over the goitrous growth and auscultation over this part may detect a bruit synchronous with the pulse. Parenchymatous changes in the gland are rare.

Exophthalmos follows the growth of the goiter in the typical case. Sometimes it appears before the goiter or without the latter. Both eyes are affected, though one may be more prominent than the other. The eyeballs gradually becoming prominent create a very distressing appearance. Sometimes the protrusion is so pronounced that the eyeball is dislocated, though in ordinary cases it rarely causes imperfection in vision. Lachrymation is often increased and conjunctivitis may be an early symptom, while the cornea may become involved in some cases. The lids are retracted (von Graefe's sign), the imperfect movement of the lid with the eyeball, is generally one of the early symptoms which is recognized by the expert. Stellwag noted the incomplete voluntary closure of the upper lids and Joffroy noted a similar imperfection in the function of the lower lids. Moebius detected a difficulty of convergence. The pupils are normal and vision is not usually perverted unless there is marked pressure.

Motor disturbances which affect the locomotion have been described, but these partake only of the general asthenia. Cramps and contractures are rare symptoms and choreic twitchings are only occasionally noticed. The *fine vibratory tremor* which attends the average case is not particularly different from that of neurasthenia.

The *vasomotor* and *secretory* disturbances are manifested by the profuse perspiration, the excessive heat of the body and occasional hemorrhages from the uterus, nose, lips and gums. Fever is not generally recognized. *Polyuria* is common and *glycosuria* is less frequent. *Albuminuria* may be detected in many cases. *Edema* may be present, but it is usually localized in the face and is not a constant symptom. The *skin,* aside from showing the copious perspiration, sometimes reveals an unnatural pigmentation distributed in irregular patches over the body. It may rarely simulate Addison's disease by the bronzing.

Digestive disturbances may be present from the beginning to the end of the disease, though they are more common as initial symptoms. Anorexia, or a voracious appetite may characterize the early stage, while vomiting, diarrhea and jaundice usually follow. During this time the patient suffers a marked emaciation, which usually continues throughout the disease. *Respiratory disturbances* may attend the exhaustion, but usually they are not marked. The *mental disturbances* are marked by periods of mental excitement, which are rarely followed by depression. The patient may be emotional and irritable, while the characteristic features of an aggravated neurasthenia are usually observed.

Diagnosis.—With the characteristic symptoms already outlined it seems almost impossible to confuse this disease with any other. Nevertheless it is many times undetected and patients afflicted with exophthalmic goiter are too frequently treated for heart disease or even phthisis. Its closest relation is with *neurasthenia,* but from this it is differentiated by its cardinal symptoms, tachycardia, goiter and exophthalmos.

Prognosis.—It may seem somewhat presumptuous if I lay claim to a favorable prognosis in the majority if not all of these cases. Despite the audacity of this statement, I fully believe that it is possible to cure nearly every case if a persistent and correct treatment is instituted. The disease may pursue a long and discouraging course, but it is certainly amenable to treatment. I have had a long and extended experience in the treatment of this disease and I have succeeded in curing many cases which have been regarded as incurable. The treatment which I shall outline should enable any patient practician to accomplish the same results.

Treatment.—All that pertains in the treatment of neurasthenia has a marked significance in the management of this disease. It is absolutely necessary to maintain complete rest, a nourishing diet and freedom from all worry or excitement. Often these precautions are all that is necessary in the early care of the case. The cardiac excitement rarely demands the use of stimulants unless there is an organic complication. *Hydrotherapy,* massage and tonics may be of adjuvant help when they are indicated, but the real treatment should be applied to the condition of the nervous system. If insomnia is a complication, we must help it temporarily by sedatives and other measures; if anemia is an underlying factor, this must receive due attention; if digestive or other disturbances contribute to the disease, these, of course, must be corrected; if the occupation or environment predispose to neurasthenic excitement, these must be changed; if a neuropathic heredity or constitutional disease are concerned, these, of course, must be taken into consideration, but the cure must be performed by the internal remedy.

Without enumeration of all the indicated remedies I prefer to mention only those which have been of paramount importance in my treatment of this disease. First of all I shall mention *lycopus virginicus* which, in its symptomatology, corresponds closely to the classical features of this disease. I have used this remedy, in my clinic and private practice, for a long time, and I am positive that it may be relied upon in the average case of exophthalmic goiter. I have experimented with the potency and the tincture and, from my experience, I am satisfied that the average case only responds to the latter. Thus it is my practice to give the tincture (m. 5-30) four times daily. The minimum dose I believe is preferable in the average case but, to control the tachycardia, the maximum dose is required in many cases. I have successfully managed some of the most intractable cases, many of which have been under the care of prominent practicians before they were sent to me, and this remedy has been my sheet anchor.

Next to lycopus I put my confidence in *fucus vesiculosus.* This has not so close a relation to the general symptomatology of the disease as lycopus, but it often is a better agent to control the cardiac activity. This, too, should be given in the tincture (m. 5-30) and it may be prescribed for a long time without any unfavorable reaction. When either of these remedies have failed me I have obtained help from the administration of *strychnia valerianate* (gr. 1/60) four times daily. I do not advise the continued use of this remedy as in the case of the other two. *Valerianate of ammonia,* in the second decimal potency, four tablets six times daily, is often a very serviceable remedy in this disease; it may be used alone or in conjunction with either of the preferential remedies mentioned above. When the nervous system is greatly excited I have successfully used the following tablet: *Zinc phosphide* (gr. 1/10), *cannabis sativa* (gr. 1/8) and *hydrobromate* of *hyoscin* (gr. 1/400), one tablet three or four times daily. In case there is an organic cardiac lesion or even when the heart excitement does not respond to the above remedies, it is necessary to further support the heart by the conjoint use of *adonis vernalis, convallaria, crategus, digitalis* or many of the other cardiac stimulants. These, however, should not be used indiscriminately nor too long.

Other remedies which have been mentioned under the subject of neurasthenia and other nervous diseases have a sphere of usefulness here, and should be employed according to their indications. The benefits of sanitarium treatment, the rest cure, massage, static electricity, vibration and all of the indicated adjuvant treatments will have their sphere of usefulness in the treatment of this disease.

ACROMEGALIA.

Acromegalia, or Marie's disease, was not recognized until 1886. It is a chronic disease characterized by gradual and usually symmetrical enlargement of the bones and soft parts of the hands, feet and face.

Etiology.—The *cause* is not yet understood, and it affects the sexes alike. It prevails between the ages of eighteen and thirty, though it may rarely appear in older patients, and congenital cases have been reported. It has been observed that patients suffering with acromegalia have been endowed with large extremities. Numerous *exciting causes* have been named, but there is no positive proof of their etiologic influence. Tabes, syringomyelia, rachitis, goiter, enlarged thymus gland and many other diseases are supposed to be closely related with this disease. The fact that the *pituitary body* is frequently hypertrophied in this disease has led to the claim that this is the cause of acromegalia. Whether it is the cause or the result of the disease is not yet known. Tumors of this body have been found without the existence of the disease. This would infer that pituitary changes have slight etiologic influence.

Pathology.—The *bones* which are affected are those of the face, cranium and extremities. In exceptional instances the bones of the trunk are hypertrophied. The frontal and occipital bones are usually thickened, the frontal and maxillary sinuses and the pituitary fossa being increased in size. The malar and inferior maxillary bones are also enlarged. The ribs, clavicles and sternum frequently show the same hypertrophy and the shoulder blades may rarely be the seat of pathologic changes. The large bones show the most decided changes at the lower ends. The metacarpals, metatarsals and phalanges are also affected. The hypertrophy affects the soft parts as well as the bones, periosteal thickening and hyperplasia also adding to the increased size of the affected parts. Pituitary gland hypertrophy is a finding in many autopsies and for this reason it has been regarded as a cause. Dana claims that the trophoneurosis is due to the defective action of this gland.

The *skin* is hypertrophied and its perverted function is often recognized by the excessive and fetid sweat. The color is slightly changed in the region of the enlargements. The patient is subject to colds, a coryza usually indicating this. The *thyroid gland* being usually atrophic, has led to the belief that this has a possible relation to the disease. The *thymus gland* is occasionally enlarged. The *kidneys* may show slight evidence of nephritis, and polyuria and glycosuria are common, while the spleen and lymphatic glands are sometimes sclerosed.

Symptoms.—The *face* gives the most conspicuous evidence of the enlargement. The large and flabby nose, the indurated eyelids, the prominent cheek bones, the enormous mouth, the thickened lips and the enlarged lower jaw form the characteristic picture of the disease. Exophthalmos is often an additional symptom, and the expression is one which excites repugnance. The *tongue* is usually large and flabby, the mucous membrane of the mouth is thickened and the teeth are often far apart. The hyperplasia also affects the soft palate, pharynx, larynx and tonsils. The *scalp* is thickened, the hair is coarse, the complexion is sallow, while the affected parts have a very unpleasant doughy feeling.

The *hands* show marked symmetrical enlargement, giving rise to the expression "spade-like." The joints are unaffected.

The *feet* present similar hypertrophic changes. "The *thorax* usually presents an anteroposterior curvature in the cervicodorsal region, which may be compensated by lumbar lordosis and attended by scoliotic twists." (Church.) The clavicular, sternal and rib enlargement cause more than normal breadth and depth of the chest, thus interfering somewhat with normal respiration. The *genitalia* may be hypertrophied or atrophied, while in women amenorrhea and sterility usually follow. *Pain* in the parts may or may not be present, severe headache being the most common complaint of this kind.

The *speech* is naturally affected and other special senses may be perverted. Vision is most frequently impaired. The muscular structure is usually unaffected; the reflexes are normal. Complications like cardiac hypertrophy, arteriosclerosis and dyspepsia are of occasional occurrence. The mentality is usually weakened with the physical feebleness.

Diagnosis.—The foregoing symptoms when present are sufficient for the diagnosis. *Myxedema* must be compared; this has no osseous deformity, the thickening of the soft parts is due to the mucin increase and the thyroid is always atrophied or absent. In *hyperostosis cranii* there is no involvement of the extremities. *Osteitis deformans* does not affect the face, while bowing of the femur and tibia may be noticed.

Prognosis.—The disease gradually progresses until there is complete physical and usually mental impairment. It may rarely assume an active form, but its chronic course may cover many years. Cardiac failure is always to be feared.

Treatment.—As yet there has been no decided advance in the therapeutic study of the disease. For this reason we are left mostly to the symptomatic treatment. Serum therapy may yet afford some help.

HYPERTROPHIC OSTEO-ARTHROPATHY.

This is a form of trophoneurosis which is characterized by a multiple osteitis which affects the terminal phalanges of both the hands and feet, the extremities of the long bones and the corresponding joints. The enlarged bony tissue shows evidence of inflammation, the cartilages are eroded and the synovial membranes are thickened. The disease is supposed to be secondary to pneumonia, but it is also probably secondary to syphilis, heart disease, spinal caries and other chronic as well as acute diseases. The joints grate on motion, the ankles, wrists and elbows are enlarged, while the deformity of the toes is often disproportionate to the deformities elsewhere. The finger ends often enlarge from the excess of subcutaneous fat. The nails then are very large and the bony tissue seems to be unaffected.

The disease evidently has a toxic origin, as it attends other diseases. It may occur at any age, is insidious in its onset and runs a protracted course. From *acromegalia* it should be distinguished by the absence of involvement of the head, face or soft parts and the particular tendency to affect the joints. The facial appearance of acromegalia is never seen in this disease.

HYPEROSTOSIS CRANII.

This disease is characterized by the marked enlargement of the bones of the cranium and, to a lesser degree, those of the face. While the inflammatory phase of the disease is evident, it is probably a trophic disorder. This is evidenced by its progressive character and the fact that it occurs with most frequency during the developmental period of life, appearing early in life or about puberty.

The *symptoms* are characterized by cranial enlargement which often succeeds inflammations like erysipelas, or rarely it follows from traumatism. *Headache,* disturbances of the cranial nerves, particularly blindness and deafness, are marked early symptoms. It has occurred in conjunction with epileptic seizures. *Facial palsy,* exophthalmos, optic neuritis and other symptoms may arise from the intra-cranial pressure. *Gradual mental enfeeblement* is usually apparent as the disease progresses. The enlargement will be recognized most decidedly in the anterior portion of the head and the upper facial bones, the bitemporal diameter being increased. The cervical vertebrae are sometimes affected. The disease responds to no treatment.

PROGRESSIVE FACIAL HEMIATROPHY.

This is a very rare disease and is characterized by a progressive atrophy on one side of the face, the skin alone being usually affected. The *etiology* is not thoroughly understood. It is considered as a neurosis and is occasionally related to other neurotic diseases like epilepsy and mental disorders. It is sometimes a feature of migraine or neuralgia. The pathology is equally unsettled.

The *symptoms* are characterized by one distinctive feature, the *flattening of the skin* on the affected side. In severe forms or when the disease has progressed, the underlying muscular and osseous tissue may display a similar atrophy. In such instances the hemiatrophy is pronounced. *White spots* are found on the affected skin where there is a loss of pigmentation; when the pigmentation is increased *brownish-yellow spots* may be observed. These changes are usually marked along the nerve-trunks, particularly the infraorbital. The hair on this side is thin, dry and frequently falls out. The sensory disturbances are not marked.

The *diagnosis* is established by the objective symptoms. In *congenital facial asymmetry* the skin over the affected side is normal and the congenital history is usually established. The rare condition of *hemi-hypertrophy* should be compared. The *prognosis* is unfavorable, though life is not endangered. The *treatment* is unsatisfactory, though electricity may be of some value in the early stage.

ANGIONEUROTIC EDEMA.

This has been known under various terms, such as circumscribed edema, giant swelling, giant urticaria, Quincke's disease, etc. It is characterized by acute circumscribed swellings of the subcutaneous or submucous tissues, the result of vasomotor disturbance rather than inflammatory irritation.

Etiology.—*Early adult life* furnishes the most patients, the average age being from twenty to thirty. Family neuroses, psychoses and degeneracy may be discovered. Often the history gives no positive information. Mental and physical exhaustion seem to be closely related to the disease. Both sexes are affected but females furnish the majority of cases. *Exciting causes* like exposure, traumatism, fright and grief are usually recognized. Tobacco, alcohol and infectious diseases are also concerned and in all probability evil habits like masturbation may predispose. The disease has a tendency to appear and disappear according to the presence of an exciting cause, cold weather most frequently inducing the acute attacks.

Symptoms.—The circumscribed swellings are of *sudden occurrence* and disappear almost as promptly. The hands, face, lips, tongue, pharynx, genitals and extremities are most frequently the parts affected. The swelling does not generally last longer than a few hours, though it may continue for several days. It may leave one locality and appear in another, though there is no symmetric or anatomic arrangement of the swellings. When it disappears it leaves no trace of its existence. Sometimes the swellings are very large, giant swellings, and again they may assume a nodular character. Tension, stiffness, burning, pricking and itching of the parts are typical symptoms. Urticarial eruptions and wheals may appear from the slightest irritation of the skin. When the larynx, pharynx, stomach or intestines are affected serious symptoms may arise. Disturbances of deglutition, digestion and respiration may be marked. Nausea and vomiting may be frequent symptoms while intestinal colic and diarrhea are not uncommon. The urine is usually increased in quantity, albuminuria and hemoglobinuria occasionally being present. Between the attacks the patient is apparently well.

The *pathology* of the disease is not known. The *diagnosis* is easily made. The *prognosis* is favorable unless the larynx becomes involved and the treatment, aside from the relief by opiates, must consider the underlying neurosis.

RAYNAUD'S DISEASE.

This disease has received its name as the result of Raynaud's description in 1862. It is a generalized trophoneurosis with a local expression of the vasomotor disturbance, the arterioles and venules undergoing attacks of spastic contraction. The fingers, toes, nose and ears will present the characteristic symptoms. Raynaud defined three stages, *local syncope, local asphyxia* and *local death,* a symmetric involvement of parts being observed.

Etiology.—The most vulnerable age is between twenty and forty, females being predisposed most frequently. Anemia and a neuropathic inheritance are concerned in its development and many of the nervous diseases are closely allied. It may also be associated with or alternate with urticaria, angioneurotic edema and scleroderma. Exposure, fright, grief, fatigue, trauma, infectious diseases and autotoxemia play an important part in its development.

Symptoms.—The *local symptoms* are characterized by the pale, glossy and bloodless appearance of the finger tips and the ends of the toes. Sometimes one finger alone may be affected and less frequently the ears, nose and lips are involved. *Loss of sensibility*, tingling and numbness of the involved parts are commonly experienced. Inasmuch as a needle prick will draw no blood the term digiti mortui has been used. These symptoms appear in paroxysms, often attended by chills and digestive disturbances which soon disappear. Later the parts are cyanotic in appearance, the results of pressure disappearing very slowly. The affected parts are rarely red, puffy and covered with perspiration. *Pain* is usually an attending symptom during the local cyanosis. Usually both hands are affected, though one may be affected later than the other. Should the local cyanosis last long, blebs may appear on the affected parts and these may end in ulceration of the part. Dry gangrene may destroy the terminal phalanges or rarely the entire finger. The mutilation of the part is, however, not proportionate to the apparent disturbance of circulation. Other parts of the body are occasionally affected.

The *constitutional symptoms* include intermittent hemoglobinuria, a decreased amount of urine and urea, and occasional attacks of uremic convulsions. Fever is not common, but cerebral disturbances may be looked for. The kidney may suffer decidedly from the angiospasm.

Diagnosis.—The typical symptoms just outlined will usually establish the diagnosis. Gangrene is recognized by its classical symptoms, while the presence of nephritis in this disease will often be a differentiating point. The decrease in eliminated urea and the presence of hemoglobinuria are marked features in favor of Raynaud's disease.

Prognosis.—The characteristic attacks of local syncope and asphyxia have a variable duration, but these return from the slightest exposure to cold, physical fatigue and nervous excitement. In older patients recovery is to be expected. In younger patients the disease may pursue a rapid course, ending in death.

Treatment.—*Local treatment* should maintain the warmth of the parts, which should be covered with cotton and other warm dressings, artificial heat being sought as far as possible. Electricity often stimulates a healthy circulation which enhances the warmth of the affected parts and some relief may be obtained by immersing the hands or feet in hot water or warm normal salt solution during the attacks. An elastic bandage has been employed to relieve the attack, refilling of the involved parts with blood after its removal often aiding the restoration of warmth to the parts. Pain may be relieved by the hypodermatic use of morphin. When gangrene ensues surgical interference may be necessary.

ACROPARESTHESIA.

This term describes a condition which may occur among washerwomen or those who are accustomed to have their hands wet. A paresthetic disturbance, often attended by pain, is experienced by such patients at night or when the weather is very cold. The fingers are cold and clumsy, while the color of the skin is either red or very white. The disorder tends to the chronic character and often becomes very resistant to treatment. The fingers and, less frequently, the toes are affected and the trouble may extend to the elbows or knees.

The diagnosis must exclude *Raynaud's disease, multiple neuritis* and *scleroderma.* The *prognosis* is favorable so far as the danger to life is concerned, but a cure is not generally attained. The *treatment* includes massage, electricity and all forms of treatment which encourage local warmth. Tonic treatment for the sake of nutrition may be indicated.

SCLERODERMA.

This is a peculiar diffuse induration and atrophy of the skin, chiefly affecting the face and extremities. It may also appear in a circumscribed form, the pathologic changes being characterized by isolated spots of induration and atrophy. These symptoms may be confined to one side or they may show a symmetrical distribution.

Etiology.—Adults are affected most frequently and females furnish the majority of cases. The discrete form may appear in early life. Traumatism, exposure and infectious diseases are considered as predisposing causes. A neuropathic inheritance, spinal and cerebral diseases are associate factors.

Symptoms.—In the generalized form pain and sensitiveness in the affected parts are common symptoms. The induration and thickness soon follows and this may be attended by redness of the parts, blebs and bullae often appearing. The skin is usually bound to the underlying tissue, giving the "hide-bound" characteristic. Atrophy follows later. The hands and face are particularly affected, the fingers are reduced in size, the bones being covered by the atrophic and glistening skin. The face shows marked evidence of skin atrophy, the bony prominences being accentuated in appearance. The epithelium is destroyed, the nails and hair also suffering atrophy. In the discrete form the induration is limited and presents sharp defining borders. Herpetic eruptions are often present. The affected spots are white and are confined to the distribution of a nerve. The face, chest, and lower extremities are usually affected most. The disease is slow in its progress, a fibrous proliferation attending the process.

Treatment.—Tonics, electricity and symptomatic treatment may give some relief, but a cure is not readily established.

LOCALIZED HYPERTROPHIES.

Symmetrical Lipomatosis.—Localized and symmetrical fatty deposits occur occasionally in adults with syphilis or alcoholism as frequent causes. These deposits are found in different parts of the body, the neck, the axilla, groins and extremities being the most common locations. Pain in these parts is sometimes present, though often there is no abnormal sensation. Neurotic symptoms are also present. Pigmentation and muscular flabbiness are characteristic symptoms. The disease slightly resembles myxedema, but the face, hands and feet are not so markedly affected. The treatment is not usually successful from a medical standpoint and surgery is often required. Thyroid feeding has been advised. The constitutional factors and the neuropathic inheritance will require attention.

Unsymmetrical Hypertrophies.—These are of rare occurrence, the neurotic inheritance being a factor. The majority of cases are congenital. A part of an extremity or one side shows the asymmetrical increase. The fatty increase is noticed in the adipocellular tissue, the muscles of these parts being impaired or rarely hypertrophic. The bones may be enlarged. Pigmentation is not common. The true nature of the disease is not known.

Chronic Hereditary Tropho-Edema.—This form of family edema appears about the time of puberty and affects chiefly the feet, legs, and rarely the entire lower limbs. It is a firm, white and painless edema. Ridlon has described a localized hypertrophy of a single finger. The disease causes little suffering and does not seem to impair the general health.

HEAT-STROKE.

As this produces such decided nervous symptoms it may be classed under nervous diseases.

Etiology.—Depreciated physical resistance always predisposes to heat-stroke when there are favorable exciting causes. Fatigue, emotional excitement, indulgence in alcohol, overeating, digestive disturbances, poor sanitation and improper food also favor its occurrence. Males are affected more frequently on account of their occupations and habits. *Sun-stroke* usually occurs among those who work hard under the exposure of the sun's rays. *Thermic fever* may appear among those who work in closely confined places during the summer months. *Heat exhaustion* is liable to occur under a variety of conditions which tend to paralyze the vasomotor centers.

Pathology.—Venous engorgement of the brain and of the cerebral and spinal membranes is observed post mortem. Rigor mortis is marked and appears early. Putrefactive changes are favored by the high temperature of the cadaver. The blood is "fluid and dark, the corpuscles are crenated and do not form rouleaux." Ecchymoses are found in the skin and serous membranes. The liver and kidneys may show parenchymatous changes. The right ventricle is generally dilated with blood and the left ventricle is rigidly contracted. The cerebrospinal system may show "an acute parenchymatous degeneration of the neurons of the whole neural axis."

Symptoms.—The *asphyxial form* of sunstroke is fortunately not common. It is characterized by *sudden dizziness,* color-blindness, throbbing headache, a lack of normal perspiration and dyspnea. A patient may fall as in a convulsion and if the heart is greatly affected death may take place suddenly. In *milder cases* nausea and vomiting take place and abdominal cramps may be pronounced. Headache is generally intense, the face is flushed, the pulse is rapid and full, the carotids and other vessels throbbing excessively, while the respiration may be stertorous. The pupils are generally contracted, the surfaces are hot and dry, and delirium may ensue. The temperature is subnormal or not higher than 102° F. A mild degree of fever may continue for some time. Fatal cases are characterized by coma, a rapid and feeble pulse with the presence of Cheyne-Stokes respiration. In favorable cases the temperature disappears by lysis, the patient gradually recovering.

The *hyperpyrexial variety* is most common. In the average case the cramp-like abdominal pains, vertigo, dizziness, nausea, vomiting and headache characterize the symptomatology. The skin is hot and dry and usually very red. The eyes are suffused and the pupils are very small. The pulse is rapid and noncompressible. Clonic spasms are often present or muscular rigidity may alternate with flaccidity. Delirium of a mild form may be present and there is often sphincter incontinence. The temperature is usually high (105°-110° F.). The respirations are increased and rarely the unconsciousness, cyanosis and dyspnea may terminate in coma and convulsions. Secondary exacerbations may appear during apparent convalescence. Pneumonia, meningitis, uremia and cardiac failure are unfavorable complications.

Heat prostration appears gradually or rarely by sudden onset, dizziness, faintness, headache, and nausea being prominent symptoms. Following the heat sensations the surface may be cold and pallid. There is muscular weakness, sighing respiration and moderate fever (100°-102° F.), the pulse, however, being rapid. Unconsciousness rarely ensues and recovery generally takes place in a few days. Nervous irritation, headache and general depression may exist for some time after apparent recovery.

Diagnosis.—The history of the case usually is a guiding feature in the diagnosis. *Acute alcoholism, meningitis, uremia* and *apoplexy* must be differentiated and these have been properly described elsewhere.

Prognosis.—Heat prostration presents a more favorable prognosis than sun-stroke. The health and habits of the patient have much to do with the outcome.

Treatment.—*Prophylaxis* is of greatest importance in the treatment, as one attack predisposes to others. Treatment of the attack will consider the severity of the seizure. In *heat prostration* the patient usually revives after being cooled off gradually and kept very quiet. It is always best to sponge the body for some time with cool water, and stimulants like *aromatic spirits of ammonia, amyl nitrate* and *strychnia* (gr. 1/60) hypodermatically, may be required. *Aconite,* or *belladonna* should be given internally. In *sun-stroke* it will be necessary to put the patient in an ice pack or sponge the parts constantly with cold water. An icebag should be applied to the head. Cardiac stimulation is often required. In *asphyxial cases* venesection, intravenous normal salt injection and external stimulation are often necessary. The feet should be put into hot water, hot water bags should be placed about the patient and ice should be kept on the head. A hypodermatic injection of nitroglycerin (gr. 1/100) or strychnia (gr. 1/60-1/20 should be immediately given. Camphor, atropin and brandy may also be given hypodermatically to overcome the shock. In case of very high temperature the ice-bath or ice-water sponge should be resorted to, but this should be done with caution lest the temperature be depressed too rapidly. The same consideration should be used regarding the administration of antipyretics. Should convulsions appear *morphin* (gr. 1/4) may be demanded hypodermatically or possibly the nervous excitement may be allayed by the internal use of *sodium bromid* (grs. 10-20) or some of the other sedatives.

When the temperature is sufficiently reduced the patient should be allowed to rest quietly in a cool place, though an ice bag should still be kept on the head. A light diet, careful nursing and the internal use of indicated remedies will be all that are essential. Diuresis must be freely established, and may demand the employment of *diuretin,* or *potassium acetate.* The patient should be given a sufficiency of water and the bowels should be regulated by saline cathartics if necessary. The convalescence must be watched with care and due attention should be paid to the susceptibility to repeated attacks at the slightest exposure.

THE INTOXICATIONS.

ALCOHOLISM.

This term is applied to acute or chronic intoxication from the drinking of alcohol. It is one of the narcomanias. It is characterized by constant or periodic craving for alcohol and is a degenerative disease.

Acute alcoholism is manifested first by a sense of warmth, exhilaration and vascular relaxation. Later functional paralysis of the nerve-centers, more or less complete, is manifested by incoördination of muscles, confusion of the faculties, and incoherent or delirious speech. Succeeding this stage acute coma, stertorous breathing, congestion of the face and a slow, full, weak pulse may be observed. The pupils may be contracted or dilated, and the eyes are congested. The patient can be aroused from his coma by shouting, by ammonia inhalations or by pressure upon the supraorbital nerve at its exit from the foramen.

The *diagnosis,* though apparently simple, is often confused with other diseases which cause a profound stupor. *Uremic coma* is most frequently mistaken for this. While the uremic convulsions may simulate those of acute alcoholism, the odor of alcohol on the breath is wanting, and in its place we may frequently detect the urinous odor. The pupil in uremic convulsions is sometimes dilated, and again contracted. The chief differentiation in the uremic state is made by the urinalysis which reveals the presence of albumin, and often casts. *Opium poisoning* is detected usually by the contracted pupils. *Cerebral hemorrhage* in an alcoholic may be diagnosed as acute alcoholism. *Diabetic coma* is detected by the ferric chloride reaction in the urine or by the finding of sugar.

Chronic Alcoholism.—This varies in degree according to the amount of alcohol imbibed, the duration of the habit, the condition of the patient and the heredity. Chronic alcoholism may be a condition of *narcomania,* which is characterized by a nervous state and a constant craving for drink, or it may take the form of a *dipsomania* in which there is a periodic desire for drink which becomes almost a mania; or *delirium tremens* often characterizes the alcoholic effects, giving rise to peculiar hallucinations, or the intoxication may assume a more profound condition of stupor and mania termed *mania a potu.*

The *heredity* or the constitutional weakness of the patient is always a predisposing factor to consider. Those who are children of alcoholic parents are more susceptible to the action of alcohol and its degenerative effects. Those afflicted with any constitutional weakness are prone to suffer from alcoholic excess more than those who are physically sound. Degeneracy furnishes a susceptibility to dementia, melancholia, epilepsy and insanity, and therefore those who inherit this degeneracy or acquire it from the use of alcohol are inclined to these diseases.

Pathology.—The pathology naturally includes the changes which are directly or indirectly related to the prolonged use of alcohol. Prominent among these may be mentioned cirrhosis of the liver, gastritis, subacute meningitis, arterial degeneration and other morbid phases which have already been described under various subjects. The direct effect of alcohol is "mainly on the protoplasm of cells, modifying or impairing their normal metabolism, at times destroying cells and substituting fibroid material, at others inciting to inflammatory action; at others simply delaying oxidation, as in the case of the adipose vesicle, whose fat remains unoxidized because its congener, alcohol, is more easily oxidized. In some instances, as in the case of the liver cells, fat is deposited in new situations because it can not be sufficiently burnt up. Different kinds of alcoholic beverages also seem to act differently, some, as gin, producing destruction of liver-cells and cirrhosis, while others, as malt liquors, produce fatty livers. It is also true that persons addicted to intermittent debauch are less liable to inflammatory lesions than constant consumers." (Tyson.) The irritation of nerve tissue from the prolonged use of alcohol not only gives rise to meningitis, but a gradual cerebritis may develop, which leads to secondary degeneration of brain tissue.

The *kidneys* also suffer decidedly from the continued use of alcohol. Contraction follows destruction of renal cells and the development of connective tissue, while these organs are also prone to enlargement as the result of fatty infiltration. We must remember that it is not only the direct action of alcohol upon these organs that does the greatest injury, but also the arterial changes, incident to the use of this intoxicant, favor the development of nephritis as a secondary disease. It must also be kept in mind that the constant stimulation induced by alcohol is liable to produce an over-functioning of all organs, to say nothing of the deleterious effects upon the mentality.

Symptoms.—The effect of chronic alcoholism is probably noticed first by the irritation of the *nervous system*. Muscular action is performed imperfectly and a constant tremor is noticed in all volitional effort. Mental deterioration is soon recognized by the sluggish intellect, though some may overcome the appearance of this weakness by a powerful will and the endurance of a good constitution. Dementia and insanity are later sequences, and these follow when the vascular degeneration leads to secondary changes in the brain tissue. Hemorrhagic pachymeningitis is a possible sequela of chronic alcoholism; multiple neuritis is almost invariably preceded by it, and general paralysis of the insane is too frequently an unfortunate sequel.

Digestive disturbances are common in this disease, being evidenced by an obstinate chronic gastritis with all its distressing symptoms, and gastric ulceration which is always serious. *Hepatic changes* in the form of hypertrophy, contraction, or fatty infiltration are prominent signs of the alcoholic effect. The portal compression, which follows from these changes, gives rise to secondary hyperemic effects in the gastro-intestinal mucosa which often resist all treatment. The *vascular* changes which arise from chronic alcoholism and secondarily affect the heart, kidneys and brain, have already been mentioned.

Delirium Tremens.—Delirium tremens or "mania a potu" is a special manifestation of chronic alcoholism due to the excessive indulgence and the prolonged action of the intoxicant on the brain. It often occurs in connection with some acute infection like pneumonia. It starts usually in the form of extreme nervous excitement, the patient being unable to sleep or keep quiet. Intense mental depression soon follows and the hallucinations of visions rapidly succeed, the patient having an idea that he is pursued by some monster or serpent. The patient may be in such a state of excitement that he trembles like a leaf, and in this state he may become unmanageable and attempt to commit suicide. Often in this stage the spirit of cunning may be the guiding influence, and unexpected actions may be looked for. The patient may complain of auditory hallucinations and be troubled with abnormal sounds. The pulse is frequent, feeble and often irregular, denoting the cardiac weakness which usually attends. There is often a temperature rise of one to three degrees and this will depend to a large extent upon the associate infectious disease.

Diagnosis.—The diagnosis in chronic alcoholism is rarely a subject of doubt if careful history is obtained. *Meningitis,* which may coexist, is differentiated as outlined under that subject. *Pneumonia* should always be looked for in a case of delirium tremens. *Apoplexy* and *uremic coma* have been mentioned under acute alcoholism.

Prognosis.—Chronic alcoholism is a curable disease, so far as the intoxication is concerned, if the alcohol can be taken from the patient. The sequelae which follow from its prolonged use are naturally incurable, though they may be relieved by an abstemious life and a perfect care of the general health. When physicians convince their patients of the danger of the alcohol habit many of our incurable chronic diseases, especially of the heart and kidneys, will not be so frequent as at present. Prosperity is often a misfortune inasmuch as it leads to an overindulgence in spirituous liquors which sooner or later leads to premature infirmity and death. Pneumonia is always a dangerous disease when it occurs in an alcoholic.

Treatment of Alcoholism.—*Acute alcoholism* requires little treatment, as the patient usually recovers after sleep has succeeded the debauch. It may in some instances be necessary to give a sedative to overcome the nervous excitement. *Chloral* (grs. 15-30) is of service in this respect. *Morphin* may be demanded to quiet an obstreperous patient, and when this is used it should be given hypodermatically (gr. 1/4), or else it serves only to irritate the patient. This drug should not be given in cases of contracted kidney, and this disease should always be suspected in cases of this kind. *Aromatic spirits of ammonia,* in dram doses, may be given in a little water. *Black coffee* is often of great assistance in sobering the patient, and if it can not be given by the mouth it may be administered per rectum. *Strychnia* (gr. 1/40-1/20) hypodermatically administered is often serviceable, as most of these patients suffer with cardiac weakness during these spells. *Vomiting* must be encouraged when there is evidence of stomach disturbance. Usually the patient does this involuntarily, but it may be encouraged by the hypodermatic use of *apomorphia* (gr. 1/15-1/10). Internal medication is rarely needed inasmuch as the patient is generally sober after a sleep. Should this be demanded *nux vomica, ignatia, ipecac, iris versicolor, cocculus* and similar remedies may be given.

Chronic alcoholism is far more difficult to treat, as it involves the absolute surrender of the habit. Thus it is essential to put the patient where alcohol can not possibly be obtained. The patient usually needs restraint, and therefore sanitarium treatment is most successful. To produce *nerve rest* is the principal object next to the withdrawal of the alcohol. In extreme cases this can be done only by the use of sedatives in sufficient dosage to enforce sleep and produce nerve calm. *Hydrobromate* of *hyoscin* (gr. 1/100) hypodermatically administered is our best single agent for this purpose. It should be given every two or three hours regularly until there is positive evidence of a subsidence in the nervous excitement; then it should be given less frequently until there is no sign of tremor or mental excitement. It is a great mistake to discontinue this remedy before the patient is absolutely tranquil. In some extreme cases this is not sufficient, and then we may add *potassium bromid* (grs. 15) and often *chloral hydrate* (grs. 15). Either of the latter remedies alone may suffice in moderate cases. *Morphin* (grs. 1/8-1/4) is rarely required in chronic alcoholism, and is a dangerous remedy in case of interstitial nephritis. *Atropin* (grs. 1/500-1/100) hypodermatically administered every three or four hours is a favorite remedy with many. *Strychnia* and *digitalis* in the usual doses are frequently required in case of cardiac weakness. Some practicians are accustomed to give an initial dose of *pilocarpin* (gr. 1/6) hypodermatically, and follow this with cardiac stimulants. It is highly essential to keep the patient in quiet surroundings during the entire period of treatment.

The general treatment which follows the specific treatment will be symptomatic, such remedies as *nux vomica, ignatia, asafetida, sumbul, arsenicum, china, agaricin, cannabis indica* and *hyoscyamus* being frequently indicated. The worst phase of alcoholism is often seen in pneumonia and this will require prompt and persistent treatment or the patient will be overcome by the extreme delirium and restlessness. In this we must supplement the remedies just mentioned with those indicated for the symptoms of pneumonia.

MORPHINISM.

The morphin habit is generally acquired from the use of this drug given for the relief of pain or insomnia. After its habitual use the patient at times suffers from an irrresistible craving for the drug, so that it may be taken in sufficient amount to cause its toxic effect. Such habitués are liable to have morphin sprees to the degree that their actions simulate those of the alcohol drunkard. Degeneracy has much to do with the habit and a neurotic tendency frequently predisposes. It is often astonishing how much morphin a patient addicted to this habit will consume without causing more than the signs of intoxication.

Symptoms.—Nervous irritability is one of the prominent symptoms of this disease, and the patient always craves the drug for its relief. A *loss of self-control* is always apparent, and with this laxity in the moral nature, *untruthfulness* becomes a fixed characteristic. The patient is always in a state of anxiety relative to some impending calamity. *Mental depression* may alternate with exhilaration, melancholia or delusional insanity frequently appearing. The withdrawal of the drug always augments these symptoms at first. The *digestion* is invariably disturbed, diarrhea taking the place of constipation in the later stages. Some morphin habitués become kleptomaniacs. *Insomnia* is usually pronounced and a *tremor* is commonly noticed in cases of long standing. *Ataxia* and *paresis* are prominent symptoms of the final stage. While death is not an early result of this habit the patient usually becomes emaciated, weak, irresponsible and susceptible to any acute disease.

Treatment.—Old cases are managed only by institutional treatment. Care and watchfulness is always necessary lest the patient, by an acquired cunning, obtain possession of the drug. When the drug is withheld some stimulant must be temporarily substituted. *Strychnia* (grs. 1/40-1/20) should be given hypodermatically every two or three hours until it is safe to reduce the dose gradually. *Aromatic spirits of ammonia* (one-half to one dram), *digitalis tincture* (m. 10), *nitroglycerin* (gr. 1/100) and *hydrobromate of hyoscin* (gr. 1/100-1/50) are serviceable as in the treatment of alcoholism. The *diet* must be nourishing, though it is often very difficult to get the patient to take enough food to sustain life.

It is invariably necessary to promote sleep by means of sedatives. *Chloral* (grs. 10-30) is probably the best remedy for this purpose. The maximum dose should not be employed when there is cardiac weakness. As morphin is a cardiac stimulant we do not frequently discover cardiac weakness in these patients. *Trional, sulphonal, somnos* or *chloralamid* (grs. 15-30) may be given with good results in many cases. The last remedy I have used successfully in much larger doses. The objection to all of these remedies is the fact that the patient soon becomes dependent upon them and this habit is often difficult to overcome. Some believe in gradually reducing the dosage of morphin in these cases. In my limited experience in these cases I have found it best to break a bad habit without any compromise.

ARSENICAL POISONING.

Acute arsenical poisoning most frequently occurs as the result of accidental or intentional poisoning by the ingestion of Paris green, "Rough on Rats" and other arsenic-containing preparations.

Symptons.—These are characterized by abdominal pain, vomiting, burning, diarrhea, rectal tenesmus, collapse and death if the dose is large. A skin eruption may be noticed, and blood and albumin may appear in the urine. Paralysis may develop later.

Treatment.—To empty the stomach is the first desideratum, mustard water or zinc sulphate (grs. 10-30) being very effective. The antidote is ferric hydrate with magnesia, or dialyzed iron, the former in 1 dram doses, and of the latter 30 minims. Lavage should also be performed until the stomach is well washed. Castor oil should be given as a purge.

Chronic Arsenical Poisoning.—This may be traced to the paper on the wall, artificial flowers and clothing fabrics. Its continued use as an internal remedy may be the cause.

Symptoms.—*Anemia* and *debility* may be the most suggestive symptoms. *Gastralgia* is usually present and a sense of burning may also affect the entire gastro-intestinal tract. *Tingling* and *pain* in the limbs may be followed by paralysis. *Peripheral neuritis* is a common development. As a later result of arsenic poisoning secondary degeneration of the epithelium in the kidneys may take place. *Muscular atrophy* often ensues in the affected muscles. Trophic changes may be noticed by the glossiness of the skin, herpetic eruptions and pemphigus. Pigmentation of the skin is sometimes observed. The extensor muscles suffer chiefly on account of the neuritis, the lower extremities being affected as much as the upper; this is a differentiating sign from lead poisoning. As the sensory nerves are also affected *pain* is a marked symptom. The symptoms and prognosis depend upon the degree of the absorption and the condition of the kidneys.

Treatment.—The patient should be removed from exposure to the drug, and symptomatic treatment should be instituted for the anemia, and the gastro-intestinal disturbances. *Potassium iodid* should be employed to aid the elimination of the poison.

LEAD POISONING.

Acute lead poisoning is not of common occurrence. Chronic lead poisoning is frequently observed. The lead may be introduced by inhalation, through the digestive tract or through the skin. It usually occurs among lead-workers and painters. Rarely the cause is found in water which passes through a lead pipe, in hair dyes and cosmetics. Tin utensils, beer, snuff and tobacco wrapped in tin foil have been factors in this condition.

Pathology.—The pathologic results of lead poisoning are widespread, the nervous system suffering most decidedly. A *toxic neuritis* is the characteristic lesion. The early changes are noticed in patches in the medullary sheath. In advanced cases there is "segmentation of the myelin and breaking up of the axis cylinder with a proliferation of the nuclei in the sheath of Schwann." Marked changes in the spinal cord are rarely found nor is the brain appreciably affected. Vascular changes are generally present and many secondary pathologic conditions may follow from these. The *musculo-spiral nerve* is chiefly involved and, following the neuritis, muscular atrophy may be discovered. The sensory nerves are not affected. Indurative nephritis is a common sequence and, with a general arteriofibrosis, may cause death.

Symptoms.—The typical *lead colic* is a marked sign of plumbism. This is characterized by sudden and intense colicky pains in the region of the umbilicus, which are twisting, grinding and paroxysmal in character. The abdomen is usually scaphoid, the recti may be rigid but tympany is not marked. Nausea and vomiting may accompany the other symptoms. Associated with this we naturally look for the *blue line on the margin of the gums. Constipation* is another marked symptom. *Anemia* due to the effect of the lead and to the secondary renal changes, loss of appetite, a fetid breath and the complaint of a sweetish taste are other features of the disease to be looked for. Microscopic examination of the blood may reveal a granular degeneration of the erythrocytes.

The *wrist-drop* is a distinctive sign of the later stage or it may be recognized in the early symptomatology: This paralysis affects the extensor muscles of the forearm though the supinator longus and the short extensor of the thumb may escape. The peroneal group in the leg may be involved. Atrophy of these muscles is often rapid. A tremor of the forearm is not uncommon. Squint may follow from ocular muscle involvement and optic neuritis is rarely present. In extreme cases epileptiform convulsions and coma may appear. Nephritis is always to be feared and gout is favored by this disease. The *cerebral symptoms* may consist of delirium and coma, which often appear with tremors and hallucinations. Aphasia, neuro-retinitis, amaurosis, headache, hysterical symptoms often bordering upon mania or melancholia and convulsions simulating those of epilepsy may be present. Insanity is not rarely a sequence of lead poisoning.

Diagnosis.—The history of typical lead colic and the characteristic blue line on the gums are usually sufficient for diagnosis in the acute stages even though the source of the lead poisoning is untraceable. The wrist-drop is the later distinctive symptom. Lead colic may closely simulate *acute appendicitis,* but in the latter disease the abdomen is usually tympanitic, there is marked tenderness to pressure over the appendix, and fever is usually present. *Pressure palsy* may cause wrist-drop but this is more frequently unilateral while the wrist-drop in lead poisoning is usually bilateral. The history is also a distinctive point in this comparison. *Acute anterior poliomyelitis* may simulate lead poisoning when the palsy in the latter affects both extremities. The former is, however, common in children while lead poisoning usually affects adults. Lead poisoning does not appear so abruptly and has little or no fever, both of which symptoms appear in acute poliomyelitis. Lead may occasionally be found in the urine if iodid of potash is given to set it free from the tissues. *Saturnine epilepsy,* epileptoid seizures caused by lead poisoning, will be distinguished from true epilepsy by the history and the attending symptoms of lead poisoning.

Prognosis.—The prognosis depends upon the degree of the toxemia and the resisting power of the patient. If the cause is removed and proper treatment instituted early, recovery should occur in the majority of cases. When cerebral symptoms appear and marked renal complications are evident a grave prognosis should be given. The paralysis depends largely upon the patient's general health and the extent of the neuritis. When the muscles have lost their reaction to electrical stimulation and particularly when the reaction of degeneration is present a cure is not to be expected though much relief may be given.

Treatment.—Any source of poisoning must be removed. Workers in lead should change their occupation. The next object is to eliminate the lead from the system as rapidly as possible. Catharsis, diuresis and skin elimination by hot baths should be sought. *Potassium iodid,* in full doses (grs. 10-30) is one of the best agents for this purpose; it should be given after meals with a full glass of water. Usually after this is given lead will appear in the urine, showing that it has been liberated from the tissues, but it frequently aggravates the symptoms. *Calcium sulphid* (gr. 2-4) every two to four hours has served me well in this respect. The general health of the patient must also be sustained by outdoor life, nourishing food, a proper amount of rest, a perfect hygiene and tonic remedies if necessary. The colic often requires *morphin* (gr. 1/4) hypodermatically, the obstinate constipation often yielding to this drug. Convulsions may respond to the hot pack while *bromids* will be indicated in extreme cases. The *paralysis* will require special attention. The faradic current should be carefully and systematically employed. *Strychnia,* in increasing doses, may be employed with success, but the iodid of potash must not be discontinued. *Ergot* has also been recommended for the paralysis.

The homœopathic remedies are very useful in this disease particularly after the colic has subsided. *Plumbum* in potency is an important remedy in the treatment of this disease and it will be useful in acute as well as chronic conditions. The colic, the blue line on the gums, the constipation and the gastro-intestinal symptoms will suggest it. *Cuprum, platinum* and *zinc* may be compared. *Nux vomica, colocynth, mercurius, camphor, cantharis* and *terebinth* are also of service during the acute stage. For the constipation and intestinal disturbances, *alumina, lycopodium, opium, sulphur, plumbum aceticum, silicea, arsenicum alb.,* and *coffea* may be used. The neuritis will demand such remedies as *agaricin, zincum pic., hyoscyamus, atropin, cicuta virosa* and *cimicifuga.*

MERCURIALISM.

Chronic mercurial poisoning may occur from the habitual ingestion of mercury or from the gradual absorption of the same in the course of an occupation. No pathologic changes have been observed except those which relate to gastric, intestinal and renal irritation. The cortex and peripheral nerves may be affected. Salivation and stomatitis have been detected as the result of its therapeutic use and chronic mercurialism has occurred from the inhalation of its vapors. Artificial teeth are rare causes and accidental mercurialization has taken place.

Symptoms.—*Stomatitis,* gastro-intestinal disturbances, salivation, ulceration of the gums, loosening of the teeth, and maxillary necrosis are common symptoms. *Anemia* and *emaciation* are results of this poisoning. The hair is often lost, the nails are brittle and skin pigmentation sometimes occurs. A characteristic *tremor* usually exists. This is at first a fine tremor which later becomes coarse and is noticed in the tongue and lips until later it appears as a general choreic spasm.

This may at times become an internal tremor or it may be associated with that of hysteria. *Aphasia,* hemianesthesia, hemiplegia and multiple neuritis are sometimes recognized. Pain in the affected muscles is often a marked symptom but atrophy and the reaction of degeneration are not usually observed. Cerebral symptoms may appear. The patient is usually very irritable and restless.

Diagnosis and Prognosis.—The characteristic symptoms usually settle the diagnosis and the prognosis is not unfavorable if the source of the poisoning is removed.

Treatment.—The cause must be removed, free elimination must be established and symptomatic treatment must be employed. *Potassium iodid* may help in the elimination of mercury while the stomatitis and salivation will be helped by the astringent solutions mentioned under those subjects.

FOOD POISONING.

Poisoning induced by infected and contaminated food is of more than common occurrence. This is no doubt due to the consumption of canned foods and preserved meats. The infection may be traced to the animal or plant which furnish the food or it may be inoculated before ingestion. The infection may also be derived from "toxicogenic bacteria and the presence of ptomains or toxalbumoses." The transmission of infectious diseases directly to man by infected meat is beyond question. We also know that food may be a medium for the direct conveyance of pathogenic micro-organisms. Saprophytic germs are also factors in the food infection.

Milk Poisoning.—This form of poisoning is particularly noticed during the summer months among children who are fed upon milk or milk products. It is, therefore, classed as a "summer diarrhea." When the milk is infected by saprophytic germs a poisonous ptomain, tyrotoxicon, is developed which creates sudden and serious symptoms. This poison has also been found in ice cream, custards, and cream puffs. The *symptoms* arising from this poison are those of gastro-intestinal inflammation, nausea, vomiting, griping abdominal pain, headache, dizziness and chilliness. Purging may follow with absolute relief of symptoms. In less favorable cases the symptoms may be so severe that exhaustion, coma and collapse may intervene, death often taking place suddenly. The *treatment* of such cases demands prompt elimination by catharsis and diuresis. If seen in time, lavage and intestinal irrigation may be of assistance. Symptomatic treatment as defined under gastro-intestinal diseases should be instituted. The heart should also be supported by cardiac stimulants.

Meat Poisoning.—Violent poisoning may take place from the ingestion of tainted meats. This is liable to result from the use of any of the canned meats and it is particularly observed when decayed sausage has been eaten. This infection applies to raw meats and cooked meats which are preserved. The *symptoms* vary. In simple poisoning a violent gastroenteritis and all of its usual manifestations will be recognized. In case of a true infection the condition will resemble that of typhoid fever. Either of these forms may terminate fatally. The prostration is always extreme and, if the patient does not die, convalescence is usually prolonged. The *treatment* consists of prophylaxis and elimination. Remedies should be given according to the symptoms, while cardiac weakness will need stimulants.

Poisoning by Fish or Shell Fish.—Ichthyismus or the intoxication which arises from the use of this kind of food is rather common. It follows most frequently from the use of canned fish. Devilled crabs, lobsters and fish salads frequently cause severe gastro-enteritis and infected oysters may cause typhoid fever or an infection similar to it.

The *symptoms* may be characterized by extreme gastro-intestinal disturbances with severe vomiting, pain, tenesmus and mucous and bloody stools. Cutaneous eruptions of an erythematous or urticarial character with extreme itching often attend. Another form is marked by cerebro-spinal disturbances, convulsions and often paralysis. The throat is very dry, the respiration is labored, dizziness is pronounced, the vision is disturbed, aphonia often ensues, ataxic symptoms are noticed, the pupils are dilated, paresis appears and the patient dies in a collapse. The *treatment* of the gastro-intestinal form will include the use of emetics, lavage, purges, enemata and remedies such as *arsenicum alb., mercurius, cuprum ars.,* etc.

GRAIN AND VEGETABLE POISONING.

Ergotismus.—This form of grain poisoning is due to the parasite claviceps purpurea, a fungus which is found in rye and other grains. Epidemics of ergot-poisoning, however, are now of rare occurrence. The nervous symptoms are marked in this poisoning. A prodromal period of weakness with headache and paresthesia may continue for several weeks before the convulsions appear. These are characterized by cramps and extreme contractures; many times they simulate epilepsy. Delirium is usually present and dementia may follow later. A form of ataxia may follow the acute poisoning.

Gangrenous ergotism is characterized by a dry gangrene which appears on the hands and feet, the fingers and toes being most affected. Formication, pain, numbness and spasm of the parts may also precede the gangrene. A line of demarcation is detected as in true gangrene, and the parts may slough. The mortality is high.

Maidismus or Pellagra.—This poisoning is the result of eating corn meal which has been contaminated. It prevails among the poorer class of people but is not common in this country. It is supposed that a bacillus causes putrefactive changes in moist corn meal with the result that ptomains develop. *Digestive disturbances,* languor, debility and restlessness are early symptoms. Very soon painful erythema with roughness of the skin appears and this is followed by an exfoliation which leaves a suppurating surface. The symptoms may suddenly assume a serious aspect, delirium and mania developing. The tendency is toward nutritional disturbance with a marked cachexia, particularly if there are repeated poisonings. The treatment should be symptomatic, prophylaxis being an important object.

Lathyrismus.—This intoxication is caused by the seed of the red German or Spanish vetch, the meal being usually mixed with that of other cereals. The symptoms are characterized by the appearance of slight stiffness of the legs which soon terminates in apparent myelitis with a resultant paraplegia. The reflexes are spastic and, in severe cases, this condition is liable to remain for some time. Death may result from the toxemia, the patient having become a paralytic.

Mushroom Poisoning.—This form of poisoning is not so common nowadays. This is due to the fact that most of the mushrooms which are now used for food are cultivated. The symptoms are nausea, diarrhea, hemoglobinemia and hemoglobinuria. In the severe intoxications convulsions may be added to the gastro-intestinal symptoms. The red agaric (amanita muscaria) is the variety of mushrooms which usually causes the poisoning and this is due to the poisonous alkaloid muscarin.

The *treatment* will demand the prompt use of *atropin* (gr. 1/150-1/100) hypodermatically and the use of *emetics* and purgatives in serious cases. Otherwise symptomatic treatment is required.

DISEASES DUE TO ANIMAL PARASITES.

NEMATODES.

Ascariasis.—Round worms (Ascaris Lumbricoides) are frequently found in the human intestine. These worms resemble the ordinary earthworm only they are provided with small papillae or hairs. They have a mouth, anus, longitudinal striae and transverse rings; there are both male and female worms, ranging from four to twelve inches in length. They inhabit the intestines of children quite frequently. The small intestine is the usual seat of their habitation, though the stomach may be occasionally invaded and, more rarely, the esophagus also. The gall-bladder has been occluded by these worms and the peritoneal cavity has been invaded by intestinal perforation. These worms usually appear in masses in the intestine and frequently cause obstruction.

The *symptoms* are not always prominent or suggestive of the real trouble. Colicky pains, anorexia, nausea, vomiting, evidence of intestinal irritation, picking at the nose and disturbed sleep are the prominent signs. Nervous children may manifest epileptiform convulsions. *Jaundice* is significant of obstruction of the bile-ducts by these worms.

The *diagnosis* is established only when the worms are found in the stools. The *prognosis* is invariably favorable.

The treatment should include strict abstinence from food for twelve or twenty-four hours or at least only a liquid diet and the use of a cathartic. *Santonin* (grs. ½-3) after the bowels have been emptied is usually sufficient. It is generally best to follow this with a good dose of castor oil. The *oil of chenopodium* (m. 5-15) administered in capsule or emulsion or on sugar, *spigelia,* fluid extract (dram 1), or *naphthalin* (grs. 3-10) are also advised. The symptomatic treatment may call for *chamomilla, cina, belladonna, nux vomica, podophyllum, iris versicolor* and similar remedies.

Oxyuris Vermicularis.—Pin worms or thread worms inhabit the rectum and occasionally the colon. Children are most frequently affected, though adults may be the host. These worms are very small, but are often present in great numbers, forming ball-like masses. The female oxyuris is about one-half inch long and the male is one-sixth inch in length. The infection takes place from the introduction of the ova through the media of water and food. The embryos mature in great numbers in the rectum.

The *symptoms* are manifested by the intense itching in the rectum, by tenesmus, and by restlessness during sleep. While the irritation is worse at night and the paroxysmal attacks generally disappear in a few days, there may be much disturbance of the nervous and digestive systems. It is possible for these worms to infest the lower colon without any marked local disturbance.

The *treatment* to be successful must destroy these worms in the rectum. For this purpose it is best to clean the rectum with a soap and water enema and then introduce the medication by injection. The decoction of quassia (ounces 1-2 of the chips to the pint of warm water), carbolic acid, tannin, vinegar, camphor and the oil of eucalyptus have been used. The injections should be introduced well up into the colon and frequently repeated to destroy the worms. The internal treatment will include a cathartic and such remedies as *cina, santonin, sulphur, nux vomica, mercurius* and *naphthalin.*

Trichina Spiralis.—Trichiniasis is due to the infection of a patient by the trichina spiralis. The male worm, when mature, measures 1/20 inch in length, while the female is twice as long. It inhabits the intestine of the hog, rat, cat and dog, as well as that of man. The embryo or muscle trichina (1/25 in. in length) is coiled up in spiral form within a capsule in the muscle sheath. When this flesh is eaten the larvae are liberated during digestion. The embryos in great numbers are set free in the stomach, where they develop rapidly and become sexually mature in five to seven days. These parasites soon pass through the wall of the intestine and reach the muscles through the lymphatics. The striated muscles are invaded, the parasites coiling themselves in the sarcolemma. They become encapsulated by the inflammatory exudate and the calcareous material which develops. The parasite may live for a long time in this capsule, but frequently it dies and calcification takes place.

Pathology.—The *pathology* is characterized by the parasitic myositis due to the invasion of the muscles by the embryos. In addition we shall discover gastro-intestinal irritation and an overgrowth of the lymph nodes in the abdominal cavity. Occasionally broncho-pneumonia, with bronchial gland enlargement, is a complication, while fatty degeneration of the liver is a rare feature of the disease. Parasitic myositis is a characteristic pathologic finding. The diaphragm, the intercostal muscles, the muscles of the neck and the abdominal muscles are usually affected. When the trichinae are numerous nearly every muscle of the body may be involved. The trichinous cysts may be observed macroscopically as grayish-white specks.

Symptoms.—*Pain* and *muscular soreness* are characteristic symptoms and may be mistaken for the so-called "muscular rheumatism." Because of the pain the patient is not inclined to move about. *Gastro-intestinal disturbances* are usually experienced before the muscle-pains are recognized; this feature pertains to the early development of the infection. About ten or fifteen days later *chills* are manifested and these are followed by a temperature (101°-104° F.) which may persist for a time. *Myositis* appears about this time. *Edema* is present in nearly every case, appearing about a week after the initial infection. The eyelids are first affected, it then extending to the other infected areas. This usually disappears after a short time, only to reappear again. Ascites is an exceptional symptom. *Edema of the larynx* and bronchial catarrh are occasionally detected. Skin disturbances such as urticaria, acne, miliaria, herpes or pruritus are occasionally manifested. Profuse sweating is often present. Insomnia and other nervous symptoms are sometimes distressing during the later stages. A marked leukocytosis is often present. Opie has called attention to the presence of eosinophilia as a diagnostic feature. This takes place about the time that the embryonal trichinae are passing from the intestine by way of the lymphatics and blood to the muscular tissue. It is therefore discovered about the third week after the initial infection. The symptoms may assume a typhoid character, headache and prostration being pronounced. *Complications* include also hypostatic pneumonia and pleurisy, while albumin and casts may be found in the urine.

Diagnosis.—The sudden facial swelling following muscular soreness, the sweating, the early gastro-intestinal disturbance, the painful and tender spots on the muscles, the fever, occasional diarrhea, and dyspnea should direct our attention to the possibility of this disease. The discovery of parasites in the pork which is used for food, and the finding of trichinae by means of a microscope in the intestinal mucus discharged from the bowel of the patient, is the most positive diagnostic evidence. The examination of a portion of the suspected muscle fiber is also a valuable method of diagnosis. *Rheumatic fever, ptomaine poisoning, cholera, typhoid fever* and *acute polymyositis* must be differentiated.

Prognosis.—This should always be guarded. Much depends upon the number of parasites ingested and the number of embryos generated in the intestines. Diarrhea is often a favorable symptom. Death is liable to occur in three to five weeks after the infection. Meat inspection is a prophylactic necessity and thorough cooking will always overcome the danger of infection.

Treatment.—Catharsis is necessary when the disease is suspected. This should be thorough and calomel is the best agent for this purpose. *Male fern, santonin* and *thymol* have been suggested as anthelmintic agents. *Picric acid* has been advised as an internal remedy to overcome the development of the encysted parasites. The other symptoms should be treated from a symptomatic standpoint. The diet should be concentrated and nourishing.

Uncinariasis (Ankylostomiasis.)—This is an infection by different varieties of worm of the uncinaria species. It is found in man and some of the lower animals. The parasite has been termed the *ankylostoma duodenale* or hook-worm. This disease is most common in the tropical and sub-tropical countries.

Etiology.—The female worm produces numerous ova and when deposited in favorable soil they develop embryos in about twenty-four hours. The embryo grows rapidly, "passing through two ecdyses" in four or five days. After the second ecdysis the embryo is infective for man. It reaches its mature form after three more ecdyses, attaining its mature form in about five weeks. Infected earth, water or food may establish the disease. The infection may take place through the skin, as children who walk barefooted in the infected soil have contracted the disease. The parasites make their way into the intestine, where the infection is developed. Earth-workers are most frequently predisposed.

Pathology.—The parasites are found in the jejunum, the mucosa of which is covered with small bloody extravasations. The bowel is thickened in spots, or there may be small cavities filled with blood containing the heads of one or two parasites. The liver and kidneys show a slight fatty degeneration. The anemia, which is the characteristic symptom, is caused by the mechanical obstruction of blood by the parasites or the "hemolytic effect from a toxin elaborated by the worm." This is detected by the increased iron in the liver, the presence of hematoidin in the liver and kidneys, and the free iron in the stools.

The *blood changes* resemble those of pernicious anemia. The hemoglobin is at first reduced, though this may increase later. Megaloblasts are present but are not so numerous as in pernicious anemia. Leukocytosis is recognized, but it is not marked. There is also a relative increase of the eosinophiles. The *feces* contain the ova and a considerable amount of blood.

Symptoms.—If there are only a few worms, the symptoms are mild. If the worms are numerous, the blood destruction is marked. In patients who are poorly nourished or in case of dysentery, dangerous complications may arise. *Colicky pains* and digestive disturbances are early symptoms. These are followed by a *progressive anemia,* with slight emaciation and edema. Food usually relieves the colic temporarily. The appetite is diminished or voracious. The anemia becomes pronounced in severe and prolonged cases. The *skin* is lemon-white in appearance, the conjunctivae and lips are pale and the sclerae are pearly. *Lassitude,* shortness of breath, vertigo and dimness of vision may follow. Crises of fever often occur, but they last only for a few days. The face and ankles are puffy, and general edema is often present. The hair of the body may fall out and malnutrition attends nearly every case.

Diagnosis.—The occupation of the patient, the anemia, the exhaustion and the appearance of triangular black or blue spots on the tongue are marked diagnostic signs. The demonstration of the ova or the parasites in the feces by microscopic test is more confirming. Stiles gives two valuable tests. A portion of the feces is placed on white blotting-paper and allowed to stand for an hour. A rusty-red discoloration develops along the edge of moisture. The second test is made by giving small doses of thymol followed by salts; the stools are collected and washed several times in a pail of water; the worms, resembling a hairpin or hatpin, with one end curved back like a hook, may be found in the sediment.

Treatment.—Thymol or its derivative, thymol urethane, is most valuable. It is given in thirty grain doses, either in capsule or in emulsion with acacia; the dose should be repeated in two or three hours. A liquid diet should be given for several days previous and the day before the drug is administered a brisk cathartic should be prescribed. After the worms have been eliminated symptomatic treatment should be applied for the anemia.

Filariasis (Filaria Sanguinis Hominis.)—The two principal varieties of filariae which afflict mankind are *filaria sanguinis hominis nocturna* and *filaria sanguinis hominis diurna.* The former is a thread-like worm, with tapering and blunt ends. The latter is slightly different in structure and is observed in the blood only during the day time. Further than this its pathologic significance is unknown. In filaria nocturna the parent worm inhabits the lymphatic system and the embryo is found in the blood. The mosquito is the intermediary host for the parasite. These filariae are discovered in the peripheral circulation of their victims only during the night. In the day time they probably enter the larger blood-vessels, particularly in the lungs. This periodicity may be changed to the day time in case the patient sleeps at that time.

The metamorphosis of the embryo to the young adult takes place in the mosquito. After a mosquito takes the blood from a patient affected with this disease, the parasites escape from their sheath when this blood reaches the insect's stomach, and they soon enter the thoracic muscles. After the metamorphosis —in six or seven days—the minute filariae migrate to the proboscis of the mosquito and are then introduced into the blood of the mosquito's victim. It is possible that the filariae may enter the water where the mosquito lays her eggs and thence into the patient's stomach. The adult parasite is a long and thin worm. "It is from three to four inches in length and the thickness of a coarse hair or bristle, and the sexes are distinct."

Pathology.—Indications of a distinct pathology are not usually discovered as the embryos are innocuous. The parent worm alone can establish a lesion. They may plug the thoracic duct or a large lymphatic trunk. As a result of this, lymphatic engorgement may take place and develop several marked symptoms. Among these we shall notice the "lymph scrotum," lymphatic groin glands, "varices of the pelvic or lumbar lymphatic trunks or of the lymphatics of the bladder, ureter or kidney." As these varicosities develop rupture may occur; if this takes place into the genito-urinary tract, chyluria is observed, or chylocele may follow if the tunica vaginalis is involved. Chylous ascites has occurred when the rupture took place into the abdominal cavity. When there is lymph stasis lymphangitis will follow, resulting in a solid edema and large tissue hypertrophies known as *elephantiasis.* Unless these marked lesions are present there may be no pathologic suggestion of the disease.

Symptoms.—*Elephantiasis* is the most marked objective symptom; this affects the legs, the scrotum of the male and the labia of the female. The breast and the arms may be rarely implicated and it may appear as pedunculated masses in different parts of the body. *Elephantoid fever* is present on account of the lymphangitis. These attacks occur at different periods. Rigors and high fever with delirium are recognized. The skin over the affected part displays a marked degree of local inflammation. The febrile attack terminates in a pronounced sweat, though the inflammatory thickening may remain. Abscesses often develop over the inflamed area. When the deeper parts are affected deep-seated pain, tenderness and signs of sepsis are present.

Hematochyluria occurs when the chyle ruptures into the genito-urinary tract. The appearance of chyle in the urine is not generally attended by other symptoms other than the presence of milky-white urine.

Varicose inguinal glands follow a varicose state of the superficial and deep inguinal glands. The swellings are doughy, soft and painless, both sides being affected. Lymph scrotum takes place when the filarial varix is located in the lymphatics of the scrotum. The testicle may be involved when the deeper glands are affected.

Treatment.—Surgery alone offers help, as the tumors must be removed. This, however, is only palliative unless the female worm is removed at the same time. No remedy has yet been discovered which will destroy the adult parasite. Local relief for the inflamed parts is necessary. If patients afflicted with this disease could be segregated so that the mosquito could not find them, dissemination of the disease would be limited.

Guinea-worm Disease (Dracontiasis.)—This parasite is termed *filaria or dracunculus medinensis* and is common in the tropics. "It is cylindric, whitish, with a blunt papillated head and a sharp curved tail. The body is nearly filled by the uterus which contains numerous embryos." The young parasite once escaping from the infected animal or man enters the intermediate host, a minute water flea (cyclops quadricornis) in which the larval development takes place. The infection occurs as the result of swallowing the cyclops, or the larva in drinking water. In this country the infection only occurs among emigrants from tropical countries. Negroes have been afflicted more than white men.

The worms enter the patient's stomach, conjugation taking place in the mesentery. The male soon dies and becomes calcified or absorbed, while the female migrates into the connective tissue. The subcutaneous tissue of the trunk, arm, leg or foot are the final lodging places. A small boil or vesicle develops, and on rupturing leaves a small sinus leading to the lodging place of the worm. The incubation period requires about one year.

Symptoms.—At the location of the parasite which is underneath a boil or vesicle, pain is experienced on pressure. When an incision is made or in case of rupture the worm may appear at the opening. A milky fluid may also exude from the wound. A slight febrile disturbance develops during the inflammatory stage of the vesicle or boil. Should the worm die it becomes encysted and is felt as a fibrous cord under the skin.

Treatment.—Prophylaxis is an essential principle of treatment. The natives in the country where this parasite is found are accustomed to pull out the worm whenever a vesicle or boil develops. Any external application which will hasten the inflammatory action will aid its expulsion. Surgery is our best aid in this respect. Emily advised the injection of a 0.1 per cent solution of mercuric chlorid into the swelling, thus causing the death of the worm.

Strongyloides Intestinalis.—The strongyloides stercoralis is a worm which infests the intestinal canal. It causes diarrhea, anemia and emacition. It is found in the tropical and subtropical countries. There are several varieties of this worm.

The *symptoms* are a continuous diarrhea, with no attending pain or temperature. As the result of the intestinal indigestion nutrition is impaired. Anemia and emaciation naturally follow.

The *treatment* will include the internal use of male fern and thymol.

Trichocephalus Dispar.—This is often called the "whipworm" and is only rarely found in the intestine of man. The male and female are nearly the same in size, the former being coiled and the latter nearly straight. The parasite is imbedded in the mucous membrane of the intestine. The cecum is the usual seat of the parasitic lesion. It does not produce serious symptoms, though diarrhea and anemia may result from its invasion.

Cestodes or Tape Worms.—These are often found in the intestines of man. They are made up of segments which are rectangular, flat and broad. They are white in appearance, each segment containing male and female organs, though they develop from the head. The head is called the "scolex" and the segment is termed the "proglottis." The head is attached to the mucous membrane of the intestine. Several varieties may be enumerated. The most common are the *tenia mediocanellata* (the unarmed tape-worm or beef-worm), the *tenia solium* (pork-worm) and the *tenia echinococcus.* The uncommon forms are the *dibothriocephalus latus,* the *tenia nana,* the *tenia confusa* and the *dipylidium caninum.* The tenia solium enters the intestinal canal as the result of eating imperfectly cooked pork; the tenia mediocanellata develops from the eating of uncooked beef and the tenia echinococcus comes from the ingestion of food which has been contaminated by the excrement of the dog.

The segments of all tape-worms discharge eggs from the intestinal canal of the host; these enter the alimentary canal of some animal, where they are hatched as parasites, which pass through the intestinal wall and then enter the muscles or other tissues, where they form cysts. When this tissue is eaten the parasites enter the intestinal canal of the victim and develop the adult worm. The *tenia solium* attains a length of several yards. The head is small and a double row of "horny hooklets" are clustered around the proboscis. Below these there are four sucking disks and by these the worm attaches itself to the mucosa of the intestine. When the egg is hatched the scolex is set free and becomes encysted in the muscle of the pig. The cysts are separated from the tissues by a connective tissue formation.

The *tenia mediocanellata* has a different head, the hooklets being absent and the four sucking disks being nearer the point of the head. The worm is much larger and the segments are broader and shorter.

The *tenia echinococcus* is not frequently found in this country. This has a double row of hooklets and four sucking disks. Only three or four segments are attached to one head, but many disconnected segments are found. The eggs enter the intestinal canal and finally wander into different parts of the body, where they form hydatid cysts. The liver is invaded most frequently, giving rise to hydatid cysts of large size on account of the developing connective tissue. Secondary or daughter cysts may develop in relation with these. On the inside of the cysts the heads of these worms develop. After a time the fluid of the cysts may be absorbed, and the scolices die, leaving a granular mass. In this mass the hooklets are found. Rarely the cysts become calcified. Irregularly distributed small cysts, the echinococcus multilocularis, walled off by connective tissue, are occasionally found in the liver. This, however, is not always due to the tenia echinococcus.

The *bothriocephalus latus* is a very large tapeworm, seldom found in this country, the segments being broad and square. The egg shaped head, which is longer than that of other tapeworms, has no hooklets or discs, being attached to the intestine by long grooves. Two species have been discovered. The infection takes place by eating imperfectly cooked fish which contain the eggs of the parasite. *Tenia nana* is a dwarf tapeworm, not exceeding two inches in length. The head has a row of hooklets and four suckers. Its intermediate host is usually the rat. The *tenia cucumerina* is a little larger than the above and has four rows of hooklets. It rarely affects man, being found in the intestine of dogs and cats. The dog-louse is the intermediate host.

Symptoms.—The presence of tape worm in man is rarely known until the segments are discovered in the stools. Gastro-intestinal irritation and a voracious appetite are suggestive symptoms. Marked nervous irritation is another symptom to be considered. A persistent anemia is often suggestive.

Treatment.—The only successful method of treatment is to abstain from food for twelve or eighteen hours and to administer remedies which will kill the worm. A thorough catharsis is necessary to expel it and unless the head is removed it will again develop. Hare advises the use of *oleoresin of aspidium* (one-half to one dram) given in capsule or emulsion. Some prefer a decoction made from the root of the male fern. *Pelletierine tannate* (grs. 6-10) is used in the same way, a purge always being given two hours later. Castor oil is a valuable cathartic when the above is given. *Pumpkin seeds* (ounces 1-3) of the crushed seeds or the resin (grs. 10-15) is also serviceable in some cases. A vermifuge made from *kamala* (drams 1-3) or *oil of turpentine* (ounces 1/2-2) in emulsion or milk have also been highly recommended.

TREMATODES.

Many of this species, which has also been described as "flukes," have been discovered in human beings.

Bilharzia disease.—This is characterized by hematuria and the formation of papillomatous tumors in the genito-urinary tract. It is caused by the schistosoma hematobium. The male and female worm are about the same size, the former forming a deep groove in which the female lies during conjugation.

This disease is limited to the African continent, only a few cases having been discovered in the United States. The ova of the worms, containing a ciliated embryo, are found in the urine. The embryos escape from the ova and move about in the urine. They soon die in the urine, but may live for a long time in water. These may enter the stomach when they exist in drinking water and develop into mature worms in the gastric or intestinal wall.

The *pathology* is characterized by the changes in the bladder; the mucous layer is covered with a bloody mucus and the epithelium shows the signs of degeneration. The submucosa is thickened. Papillomas of various sizes are found in the bladder, the submucosa contains numerous ova, many of which have undergone the process of calcification. The rectum, urethra, ureters, prostate or uterus may partake of these pathologic changes; thus stricture, urinary fistulae, urethritis, pyelitis and prostatitis are frequently attending complications. Malignant bladder disease or nephrolithiasis are often terminal diseases.

The *symptoms* depend upon the intensity of the infection and the extent of the lesion. *Hematuria* is the most conspicuous symptom, the blood in the urine often being excessive. In the more serious cases *cystitis* is present and the signs of a bladder tumor or stone may be detected. Should suppuration ensue fistulous tracts may develop. When the bowel is affected *dysentery* may take the place of the hematuria.

The *blood changes* are often marked, a decrease of red blood cells and a diminished hemoglobin percentage often being noticed. This may point to a toxic or parasitic anemia.

The *prognosis* depends upon the extent of the infection and the number of the worms present. If reinfection does not take place the adult worms usually die and the ova are evacuated. The convalescent period, however, may extend over several years.

Treatment.—No satisfactory specific treatment has been discovered. The most that can be done is to treat the disease according to the symptomatic indications. Prophylaxis is, of course, an important feature for consideration.

Distomiasis of the Lung.—This disease has also been referred to as *lung flukes* or *parasitic hemoptysis,* and it is observed only in Oriental countries. The parasite is a small fleshy worm, or fluke, and is generally found in the lungs. These worms discharge numerous ova, which are found in the sputum. The infection is probably developed through the medium of contaminated water.

The autopsy reveals hemorrhagic infarcts in the lungs and these patches are infiltrated with cavities which contain one or two distoma and masses of eggs.

The *symptoms* are characterized by the cough, with bloody or prune-juice sputum. The hemoptysis may at times be very severe and at other times the sputum has a rusty color, due to the blood and the brown colored ova. The disease usually becomes chronic and continues for several years. The hemorrhage and secondary anemia are the unfavorable symptoms. No *treatment* has as yet given satisfaction.

Distomiasis of the Liver.—Liver flukes are prevalent in Japan in epidemics; emigration has contributed the disease to other countries, but it has made no permanent settlement in these localities. The parasite enters the biliary tract or the pancreatic duct and it may also be found in the duodenum, the stomach and spleen. The obstruction of the biliary ducts causes hepatic catarrh and an increase of connective tissue; there may be a slight hemorrhage from the biliary tract and anemia is not an uncommon secondary result. Cyst-like cavities are often formed in the gall-bladder, where the parasites are found in great numbers.

The *symptoms* are characterized by an abnormal appetite and a marked enlargement of the liver. Diarrhea and impaired nutrition follow later, the patient becoming very weak and emaciated. Dropsy soon ensues and then recovery is impossible.

TRYPANOSOMIASIS.

Infection caused by trypanosomata occurs in two forms, the trypanosoma fever and sleeping sickness (African lethargy). The parasites which cause these diseases are flagellated protozoa and there are several varieties. Animals are infected by ingestion of the parasites into the stomach. The infection may be carried by water, fleas, lice, mosquitoes and ticks. Flies are the most probable disseminators of the disease.

Trypanosomia Fever (Human Trypanosomiasis).—The symptoms of this infection are characterized by the remittent fever, the rapid pulse, marked weakness, edema of the face and enlarged spleen. The parasites may exist in the blood without any pronounced symptoms. An apyretic period usually alternates with the fever. Quinin does not affect the fever. Erythematous patches may appear on the body and the edema is associated with these. The lymph nodes are enlarged and the parasites may be found in these as well as in the peripheral blood. There is a moderate anemia and the large mononuclear leukocytes are increased.

The treatment is not satisfactory, though *arsenic, sodium cacodylate,* or *arsenate of iron* have been given hypodermatically. *Methylene blue* and *quinin* have apparently failed as therapeutic agents. The patient must be kept away from the infected flies.

Sleeping Sickness (African Lethargy).—This is a disease peculiar to tropical Africa. It is caused by the entrance of the trypanosoma into the cerebrospinal fluid or the blood. The disease appears most commonly among the negroes. The lesion is probably an extensive meningo-encephalo-myelitis.

The *symptoms* are characterized by a long incubation period, psychical prodromata announcing the attack. Headache, vertigo, facial puffiness and slight fever are in evidence before the lethargy begins. Somnolence is an early feature, the patient staggers during the waking moments if he attempts to walk and then falls into a deep sleep. Nutritional disturbance naturally follows and the patient is greatly emaciated. Paralysis may be a terminating symptom or coma may supervene.

Treatment is not very successful. Purging is of some help and large doses of arsenic have been recommended.

Psorospermiasis.—This refers to a condition arising from the development of psorosperms into parasites. These produce nodules, which may be felt through the abdomen. The symptoms of typhoid fever are recognized, diarrhea, stupor, slight fever and hepatic tenderness being usually present. Autopsy reveals tuberculous-like masses in the liver, spleen, peritoneum and kidneys. Cases have been observed in which tuberculosis of the skin was the final termination, the lymphatics and lungs become affected secondarily.

DHOBIE ITCH.

This term is applied to a group of itching skin diseases which are common in the warm climates. Three types of the disease are recognized. The first is due to the *microsporon minutissimum,* the second class represents the various types of *tricophyton* or ring worms, while the third type is the dhobie itch or *pemphigus contagiosus,* due to a diplococcus. This class of diseases has been imported to us from the tropics. The disease always spreads by contact and the clothing which is washed in cold water is the usual means of its spreading. It may be obtained by sexual intercourse and bathing in contaminated waters is a common cause of its dissemination.

The *lesion* is found in the parts covered with hair like the axilla and groin. In appearance the lesion resembles a ring worm, with scaly margins. The interior of the patch is red, glossy and bare. Secondary infection usually starts from scratching. *Smarting* and *itching* are severe and heroic treatment is required to obtain relief. Improvement or a cure take place when cold weather arrives.

The *pemphigus contagiosus* appears in the form of vesicles or blebs. When these break a red and raw surface is exposed. It may simulate varicella during its early stages, but constitutional symptoms are not present. The disease is seen principally in children.

The *treatment* will include the thorough cleansing of the parts with green soap and the application of a parasiticide. Vleminck's solution of the *sulphuret* of *calcium* has been successfully applied at night. *Iodin tincture* painted over the parts daily and an ointment of *chrysophanic acid* or the *oleate of mercury* have been advised. A saturated solution of salicylic acid in collodium applied twice daily is recommended by Hare. Reinfection must be avoided; the underclothing must be carefully boiled and the patient should avoid all other possibilities of contamination.

Chiggers (Sand Flea).—This insect is most common in the tropics. It is a very small reddish-brown flea, which attaches itself to the surface of the skin. The female, when impregnated, burrows under the skin. When the ova are mature, if undisturbed, they are expelled at the point of entrance. Chiggers may inflict a patient in large numbers, the exposed parts being subject to the infection. Following the bite of the insect there is intolerable itching, papules being formed, and these soon terminate in pustules, ulcers form from these and healing leave scars. Extensive infection and sloughing may take place when asepsis is not observed.

The *treatment* must include complete enucleation of the pustule to get rid of the parasite. The essential oils, turpentine, infusion of tobacco and mercurial ointments have been used for the itching.

MYIASIS.

This infection may occur from the larva of the common blue bottle-fly. The eggs which are deposited on the body hatch into larvae in a few hours. Circles of minute spines cover the body of the worm, hence the term screw-worm. The larvae burrow extensively, frequently causing much tissue destruction. On account of their activity and the circles of spines they are not easily removed when found. The larvae may enter the nose and work their way into the ethmoidal or frontal sinuses or meninges. They may also enter the tympanic cavity by perforating through the ear.

The *treatment* consists in the injection of strong parasiticides into the affected parts. Carbolic acid solution and chloroform have been successfully used. It is best to apply these directly on the parasites when they may be easily extracted.

Intestinal myiasis occurs as the result of the ingestion of fly-blown food. Many species of infection have been observed. The existence of the larvae is usually unknown until they are found in the stools. Severe gastro-intestinal symptoms are the exception. The disease is most frequent in tropical countries. A free purge usually removes the larvae from the bowels.

Dermatobia cyaniventris arises from the infection caused by the deposit of eggs, of the common American fly, upon the skin of man and cattle. The cutaneous tissue is penetrated by the larvae, pustular lesions resulting.

INDEX.

Abscess of brain 970
  of liver 766
  of lungs 441
  paranephric 871
  retropharyngeal 611
Acids of digestion 628
Acetonuria 819
Acute pernicious multiple-neuritis 886
Acromegalia 1048
Acroparesthesia 1055
Actinomycosis 258
  bacteriology of 258
  diagnosis of 260
  pathology of 259
  symptoms of 259
  treatment of 260
Addison's disease 368
  diagnosis of 370
  etiology of 369
  pathology of 369
  prognosis of 370
  symptoms of 369, 370
  treatment of 371
Adenitis tuberculous 204
Albuminuria 816
Albumosuria 817
Alcoholism 1060
  chronic 1060
Amyloid degeneration of kidney 826
  of liver 763
  of spleen 791
Amyotrophic lateral sclerosis 953
Anemia 342
  cerebral 969
  diagnosis of 344
  due to hemorrhage 342
  due to inanition 343
  due to malignant disease 342
  due to drain of chronic disease 343
  due to toxins 343
  infantum pseudo-leukemia 367
    symptoms of 367
    treatment of 368
  primary or essential 345
  prognosis of 344
  progressive pernicious 350
    blood examination in 352
    diagnosis of 353
    etiology of 350
    pathology of 350
    prognosis of 354
    symptoms of 351, 352
    treatment of 354
  symptomatic or secondary 342
  symptoms 343
  splenic 365
    diagnosis and prognosis of 366
    pathology of 365
    treatment of 367
    symptoms 366
  treatment 344
  chlorotic 345
    blood changes in 347
    diagnosis of 348
    etiology of 345
    pathology of 346
    prognosis of 348
    symptoms of 346
    treatment of 348
Aneurysm 579
  arterio-venous 579
  cardiac 555
  dissecting 579
  false 579
  miliary 579
  of abdominal aorta 586
  of heart 555
  of pulmonary artery 588
  of thoracic aorta 580
Angina Ludovici 599
  pectoris 567
  pseudo-angina, differential points of 570
  treatment of 571
Angioneurotic edema 1052
Ankylostomiasis 1078
Anorexia 680
Anosmia 894
Anthrax 260
  diagnosis 262
  etiology of 260
  external 260
  internal 261
  prognosis of 262
  symptoms of 260
  treatment of 262
Anthracosis 443
Aortic insufficiency 505
  regurgitation 505

    - diagnosis 508
    - pathology 505
    - physical signs 507
    - symptoms of 506
      - Corrigan's pulse in 507
  - stenosis 509
    - diagnosis of 511
    - pathology of 510
    - physical signs 511
    - symptoms of 510
  - aortic regurgitation, and mitral regurgitation 520
  - aortic stenosis with regurgitation 521
- Aphasia 982
  - auditory 982
  - graphic-motor 983
  - motor 982
  - visual 983
- Aphemia 982
- Apoplexy pulmonary 429
- Appendicitis 732
  - diagnosis of 735
  - etiology of 732
  - pathology of 733
  - symptoms of 733
  - treatment of 738
- Argyll-Robertson pupil 937
- Arrhythmia, cardiac 565
- Arsenical poisoning 1066
- Arteriosclerosis 574
  - diffuse 575
  - circumscribed 575
  - symptoms 575
  - treatment of 577
- Arthritis deformans 300
  - and gout, differential points in 304
  - bacteriology of 301
  - diagnosis of 304
  - etiology of 300
  - Heberden's nodes in 303
  - pathology of 301
  - predisposing causes of 301
  - prognosis of 304
  - symptoms of 302
  - treatment of 304
  - varieties of 303
- Ascariasis 1075
- Ascites 801
- Asthma 414
  - diagnosis of 416
  - etiology of 414
  - hay (see *autumnal catarrh*) 385
  - spasmodic 904
  - symptoms of 415
  - treatment of 417
- Ataxia, Friedreich's 945
  - locomotor 934
- Atelectasis, pulmonary 432
- Athetosis 1035
- Atrophy, acute yellow, of liver 769
  - progressive muscular 951
  - spastic progressive muscular atrophy 953
- Autumnal catarrh 385
- Bacelli's sign in empyema 461
- Bacilluria, typhoid 20
- Bacillus anthraçis 260
  - diphtheriae 128
  - dysenteriae 67
  - icteroides 81
  - influenzae 171
  - leprae 254
  - mallei 257
  - of Eberth 1
  - of Klebs-Loeffler 128
  - of Pfeiffer 171
  - of tuberculosis 197
    - hereditary transmission of 198
    - inhalation of 198
    - sources of 197
    - swallowing of 198
  - of the plague 91
  - proteus fluorescens 276
  - Xerosis 129
- Bacteriology, of actinomycosis 258
  - of rheumatic fever 116
  - of anthrax 260
  - of appendicitis 732
  - of arthritis deformans 301
  - of cerebro-spinal meningitis 93
  - of cholera 75
  - of chronic amebic dysentery 69
  - of dengue 88
  - of dyspeptic enteritis acute 694
  - of diphtheria 129
  - of dysentery 67
  - of epidemic cholera 75
  - of erysipelas 103
  - of gonorrheal arthritis 127
  - of glanders 257
  - of hydrophobia 263
  - of influenza 171
  - of leprosy 254
  - of lobar pneumonia 178
  - of malarial fever 51
  - of Malta fever 49
  - of measles 158
  - of mumps 169
  - of paratyphoid fever 39
  - of pericarditis 473, 483
  - of plague 91
  - of pleurisy, sero-fibinous 450
  - of progressive pernicious anemia 350
  - of pyemia 112
  - of relapsing fever 46
  - of scarlet fever 149
  - of scorbutus 326
  - of septicemia 109
  - of syphilis 241
  - of tetanus 266
  - of tuberculosis 196

    of typhoid fever ... 1
    of ulcerative endocarditis .. 493
    of variola ... 138
    of Weil's disease ... 276
    of yellow fever ... 81
Banti's disease ... 367
Basedow's disease ...1043
Bednar's aphthae ... 594
Bell's palsy ... 899
Beri-beri ... 884
Beta-oxybutyricaciduria ... 819
Big-jaw (see *actinomycosis*) ... 258
Bile-ducts, carcinoma ... 753
    stenosis of ... 754
Bile-tract, diseases of ... 741
Bilharzia disease ...1085
Biliary calculi ... 749
Black death (see *plague*)... 91
Black smallpox ... 141
Bladder, hemorrhage of... 880
    irritability of ... 880
    neoplasms of ... 879
    stone in ... 879
Blood in appendicitis ... 735
    in diabetes ... 285
    in diphtheria ... 131
    in leukemia ...356, 357, 358
    in pseudo-leukemia ... 362
    in typhoid fever ... 10
Boas' resorcin test of gastric
        contents ... 630
Brachial plexus, diseases of 908
Brachycardia ... 564
Bradycardia ... 564
Brain, abscess of ... 970
    anemia of ... 969
    hyperemia of ... 969
    tumors of ... 985
Break-bone fever (see *dengue*).. 87
Bright's disease, acute... 834
    chronic ... 846
Bronchi, diseases of ... 400
Bronchial asthma ... 414
    stenosis ... 413
Bronchiectasis ... 409
Bronchitis, acute ... 400
    diagnosis of ... 402
    etiology of ... 401
    pathology of ... 401
    prognosis of ... 402
    symptoms of ... 401
    treatment of ... 402
  capillary ... 432
  catarrhal ... 400
  chronic ... 404
    diagnosis of ... 406
    etiology of ... 404
    pathology of ... 404
    prognosis of ... 406
    symptoms of ... 405
    treatment of ... 406
    varieties of ... 405
  fetid ... 406
  fibrinous ... 420
  in pneumonia ... 185
Bronchocele (see *goiter*) ... 372
Broncho-pneumonia ... 432
    diagnosis of ... 433
    etiology of ... 432
    pathology of ... 432
    prognosis of ... 433
    symptoms of ... 432
    treatment of ... 433
Broncho-pulmonary hemorrhage. 426
Bronchorrhea ... 405
Brown-Séquard's spinal paralysis 959
Bubo, parotid ... 601
Bulbar paralysis ... 954
Bubonic plague (see *plague*).... 91
Buhl's disease ... 336
Cachexia strumipriva ... 376
Caisson disease ... 914
Calcareous degeneration of the
        heart ... 552
Calculi biliary ... 749
    renal ... 828
Calculus of pancreas ... 790
Cancrum oris ... 549
Carcinoma ... 723
    of bile-ducts ... 753
    of esophagus ... 622
    of intestine ... 722
    of kidney ... 873
    of liver ... 772
    of lung ... 445
    of pancreas ... 786
    of peritoneum ... 801
    of stomach (see *gastric carcinoma*) ... 659
Cardiac aneurysm ... 555
    dilatation ...540
    disease, combined valvular.. 518
    hypertrophy ... 533
Cardia, insufficiency of... 679
Cardialgia ... 665
Cardiospasm ... 679
Carditis (see *myocarditis*)... 544
Catarrh, acute gastro-intestinal.. 691
    autumnal ... 385
    nasal, acute ... 381
    chronic ... 383
Catarrhal bronchitis ... 400
    enteritis, acute ... 683
      chronic ... 688
    pneumonia ... 432
Cavities in chronic tuberculosis.. 213
Celiac disease ... 701
Cerebral hemorrhage ... 974
Cerebral palsies of children... 483
Cephalodynia ... 271
Cerebro-spinal meningitis ... 93
    anomolous, forms of ... 97
    arthritis in ... 97
    Babinski, reflex in ... 97

  - bacteriology of 94
  - complications in 97
  - diagnosis of 98
  - etiology of 93
  - lumbar puncture in 101
  - leukocytosis in 96
  - pathology of 94
  - prognosis of 99
  - symptoms of 95
    - cutaneous 96
    - gastro-intestinal 95
    - Kernig's sign 97
    - nervous 95
    - renal 97
  - treatment of 100
- Cervical plexus 908
- Cestodes 1083
- Chalicosis 443
- Chickenpox (see *varicella*) 147
- Chills and fever (see *malarial fever*) 50
- Chiggers 1089
- Chloroma 368
- Chlorosis (see *anemia*) 345
- Choked disk 895
- Cholecystitis, acute infectious 747
  - calculous 749
- Cholelithiasis 775
- Cholera 75
  - diagnosis of 78
  - etiology of 75
  - pathology of 76
  - prognosis of 79
  - symptoms of 77
  - treatment of 79
  - infantum 698
  - morbus 691
  - nostras 691
  - sporadic 691
    - treatment of 693
- Cholerine 78
- Choluria 818
- Chorea 1024
  - electrica 1033
  - fibrillary 1033
  - gravis 1027
  - Huntingdon's 1032
  - paralytic 1028
  - of pregnancy 1027
  - rhythmic 1033
  - Sydenham's 1024
  - with hemiplegia 1035
  - treatment of 1028
- Choreiform affections 1033
- Chyluria 818
- Chvostek's sign 1039
- Cirrhosis of the liver 777
  - of lung 430
- Climatic treatment of tuberculosis 237
- Coal-miner's disease 443
- Coin-test in pneumothorax 466
- Colic, mucous 798
  - renal 829
- Coccygodynia 893
- Colitis 683
- Coma, diabetic 287
- Common duct, obstruction of 750
- Constipation 714
- Consumption, galloping 210
- Cord, spinal, syphilis of 245
  - tumor of 958
- Coronary arteries, diseases of 575, 568
- Coryza, acute (see *acute rhinitis*) 381
- Cowpox 147
- Creosote in treatment of tuberculosis 236
- Cranial nerves, diseases of 894
- Cretinism (see *myxedema*) 378
- Croup (see *diphtheria*) 128
  - false (see *spasmodic laryngitis*) 393
- Croupous pneumonia (see *lobar pneumonia*) 178
- Crutch-palsy 908
- Cycloplegia 896
- Cyst, hydatid, of lung 446
  - of pancreas 788
  - renal 872
- Cystic duct, obstruction of 750
  - kidney 872
- Cystinuria 822
- Cystitis 875
- Day-blindness 895
- Degeneration, amyloid of heart 532
  - of liver 762
  - of spleen 791
- Delirium tremens 1062
- Dementia paralytica 990
- Dengue 87
  - complications of 89
  - diagnosis of 89
  - etiology of 88
  - symptoms of 88
  - prognosis of 89
  - treatment of 89
- Dhobie itch 1085
- Diabetes insipidus 297
  - diagnosis of 298
  - etiology of 297
  - pathology of 297
  - prognosis of 298
  - symptoms of 297
  - treatment of 298
- Diabetes mellitus 282
  - blood in 285
  - coma in 287
  - diacetic acid in 286
  - diagnosis of 288
  - etiology of 284
  - kidneys in 285
  - nervous system in 282, 285
  - pancreas as causative factor of 282, 285

- pathology of 285
- Pavy's theory of 284
- peripheral neuritis in 288
- prognosis of 289
- role of liver in 283
- suprarenal glands in 283
- symptoms of 286
  - cutaneous 286, 287
  - digestive 287
  - muscular 288
  - nervous 287, 288
  - respiratory 287
  - urinary 286
- traumatism in 283, 284
- treatment of 289
  - dietetic 289
  - medicinal 292-295

Diacetic acid in urine 286, 819

Diaceticaciduria 819

Distomiasis, of lungs 1086
- of liver 1087

Diarrhea, nervous 729
- summer 691

Diazo-reaction of typhoid fever 24

Dietl's crises 807

Dilatation of the heart 540
- of stomach 635

Diphtheria 128
- and follicular tonsilitis, differential points of 135
- bacteriology of 129, 131
- blood in 131
- cervical lymphatics in 131
- complications in 131-134
- diagnosis of 134
- etiology of 129
- heart in 131-134
- immunity to 130
- kidneys in 131
- laryngeal 133
- lungs in 131
- malignant 132
- modes of infection in 129
- nasal 133
- nerves in 131
- pathology of 130
- pharyngeal 132
- prognosis of 135
- simple tonsillar 132
- site of infection 129
- symptoms of 131, 132
- treatment of 136
  - antitoxin 136
  - local 137
  - medicinal 138
  - prophylactic 136
- wound 134

Diplegia 983

Diplococcus meningitidis 94
- scarlatinae 149

Diplopia 896

Distoma hematobium 1085
- hepaticum 1087
- pulmonale 1086

Distomiasis 1086

Diverticulum, esophageal 620

Dracontiasis 1082

Dracunculus medinensis 1082

Dropsy of pericardium 486
- of peritoneum 801
- of pleura 449
- cardiac, treatment of 531
- of renal disease 836

Ductless glands, diseases of 368

Duodenitis 683

Dura mater, inflammation of 916

Dysentery 66
- amebic 69
  - pathology of 69
  - symptoms of 70
  - complications of 70
- bacillary form 67
  - bacteriologic diagnosis 68
  - pathology of 68
  - symptoms of 68
  - complications of 69
- catarrhal 66
  - etiology of 66
  - pathology of 66
  - duration of 67
  - diagnosis of 67
  - symptoms of 67
- chronic 70
- general treatment of 71

Dyspepsia 639

Eberth's bacillus 2

Edema, angioneurotic 1052
- collateral 424
- general pulmonary 424
- of lungs 424
- pulmonary 424

Ehrlich's reaction in typhoid fever 20, 24

Eighth nerve 900

Electrolysis in aneurysm 586

Elephantiasis 1081

Eleventh nerve 905

Embolism of brain 979
- of portal vein 757
- pulmonary 429

Embryocardia 565

Emphysema 435
- compensating 435
- hypertrophic 436
  - diagnosis of 438
  - etiology of 436
  - pathology of 436
  - physical signs of 438
  - prognosis of 439
  - symptoms of 437
  - treatment of 439
- interlobular 435
- senile 439
- vesicular 435

Empyema 460
  Bacelli's sign in 461
  etiology of 460
  of pericardium 483
  pathology of 460
  symptoms of 460
  treatment of 462
Encephalitis, acute 972
  exudative with hemorrhage. 973
Endarteritis deformans 574
  obliterating 574
Endocarditis 488
  chronic 496
  fetal 489
  malignant 493
  sclerotic 496
  simple acute 488
  ulcerative 493
  valvular 488
Enteralgia 728
Enteric fever (see *typhoid fever*) 1
Enteritis, acute catarrhal 683
  symptoms of 684
  treatment of 685
  chronic catarrhal 688
   symptoms of 689
   treatment of 690
  pseudo-membraneous 702
  phlegmonous 702
Enteroptosis 682
Enterocolitis, acute 697
Enterospasm 730
Enuresis 881
Ephemeral fever (see *febricula*) 277
Epidemic catarrhal fever (see *influenza*) 171
  cholera 78
  hemoglobinuria, of new-born 335
  parotitis (see *parotitis*) 169
Epilepsy 994
  Jacksonian 999
  nocturnal 998
  petit mal 998
  procursive 998
  psychic form 998
  psychic, epileptic equivalent. 999
  symptoms of 996
Epistaxis 387
  in typhoid fever 11
Erb's sign 1039
Ergotismus 1073
Erysipelas 103
  bacteriology of 103
  complications of 106
  diagnosis of 106
  etiology of 103
  pathology of 104
  prognosis of 107
  sequelae of 106
  symptoms of 104
  treatment of 107
   local 107
   constitutional 108
Erythromelalgia 893
Esophageal diverticulum 620
Esophagismus 616
Esophagitis, acute 615
Esophagus, diseases of 614
  carcinoma of 622
  dilatation of 620
  diverticula of 620
  spasm of 616
  paralysis of 618
  stricture of 618
  ulcer of 621
Estivo-autumnal fever (see *malarial fever*) 57
  ameba of 53
Ewald test-breakfast 627
Exophthalmic goiter 1043
Eye, paralysis of muscles of 896
Facial nerve 898
Fallopian tubes, tuberculosis of 231
False croup (see *spasmodic laryngitis*) 393
Family periodic paralysis 1042
Farcy (see *glanders*) 256
Fatty liver 759
Febricula 277
Fecal obstruction 709
Fetal endocarditis 489
Fever and ague (see *malarial fever*) 50
  aphthous (see *foot and mouth disease*) 280
  catarrhal (see *influenza*) 171
  ephemeral (see *febricula*) 277
  glandular 281
  hay (see *autumnal catarrh*). 385
  in chronic tuberculosis 217
  in tetanus 268
  malarial 54
  Malta 49
  miliary 279
  mountain 40
  paratyphoid 39
  relapsing 46
  rheumatic 115
  scarlet 149
  simple continued (see *febricula*) 277
  splenic (see *anthrax*) 260
  thermic 1057
  typhoid 1
  typhus 40
  yellow 81
Fibrinous pneumonia 223
Fibroid phthisis 223
Fiedler's disease (see *Weil's disease*) 276
Fifth nerve 897
Filaria medinensis 1092
  sanguinis hominis 1080
Filariasis 1080

Floating kidney 806
  spleen 790
Flukes 1088
Food poisoning 1071
Foot and mouth disease 280
Fourth nerve 896
French measles (see *Rubella*) 162
Friedreich's ataxia 945
Gall-bladder, in typhoid fever 17
Gall-stones 749
Galloping consumption (see *acute phthisis*) 210
Gallop rhythm 566
Gangrene of lungs 440
Gastralgia 665
  treatment of 667
Gastrectasis 635
  diagnosis of 638
  physical signs 637
  treatment of 638
Gastric contents, examination of. 627
  ulceration 653
    diagnosis of 655
    etiology of 653
    hematemesis in 655
    pathology of 654
    symptoms of 654
    treatment of 656
Gastritis, acute catarrhal 639
  symptoms of 640
  treatment of 641
  acute toxic 642
    symptoms 642
    treatment of 643
  chronic catarrhal 644
    diagnosis of 647
    etiology of 644
    pathology of 645
    prognosis of 647
    symptoms of 646
    treatment of 647
  gastric ulcer and gastric carcinoma, differentiated 651-661
  phlegmonous 644
Gastric carcinoma 658
  diagnosis 661
  pathology of 659
  symptoms 659
Gastric neuroses of motility 678
Gastrodynia 665
Gastro-enteritis 691
Gastroptosis 633
Gastro-succorrhea 679
Genito-urinary organs in tuberculosis 231
German measles (see *rubella*) 162
Giantism 1048
Gin-drinker's liver 777
Glanders 256
  acute 257
  chronic 257
Glandular fever 281
Glassblower's mouth 601
Glenard's disease 682
Glomerulo-nephritis (see *acute nephritis*) 834
Glossitis 597
  acute 597
  chronic superficial 598
  dessicans 598
Glosso-pharyngeal nerve 903
Glosso-labio-laryngeal paralysis. 954
Glycosuria 982
  alimentary 983
Goiter 372
  diagnosis of 374
  etiology of 372
  pathology of 373
  symptoms of 373
  treatment of 374
  exophthalmic 1043
    diagnosis of 1046
    etiology of 1044
    prognosis 1046
    symptoms of 1044
    treatment of 1046
Gonorrheal arthritis 127
Gout 306
  alcohol in 307-309
  acute 309
  arthritis deformans, differential points 311
  chronic 309
  diagnosis of 311
  diet in 311
  etiology of 307
  heart and bloodvessels in 308
  heredity in 307
  irregular 310
    symptoms of 309
  kidneys in 310
  pathology of 808
  treatment of 311-315
  urinary symptoms in 310
Grand mal 996
Gravel 828
Graves' disease 1043
Green sickness (see *chlorosis*) 345
Grippe (see *influenza*) 171
Guinea-worm disease 1082
Günzburg's test 629
Habit spasm 1035
Hay asthma (see *autumnal catarrh*) 385
  fever 385
Heart, aneurysm of 555
  atrophy of 555
  brown atrophy of 552
  congenital affections 572
  amyloid 552
  calcareous 552
  fatty 552
  infiltration 552
  degeneration 552

dilatation of 540
symptoms of 541
treatment of 543
hypertrophy of 533
diagnosis of 537
etiology of 534
pathology of 534
physical signs of 536
prognosis 538
symptoms of 535
treatment of 538
neuroses of 557
palpitation of 557
rupture of 556
Heat exhaustion 1057
treatment of 1059
Heat stroke 1057
Heat prostration 1058
Heberden's nodosities 303
Hematemesis 663
Hematochyluria 1081
Hematomyelia 912
Hematuria 813
Hemeralopia 895
Hemiatrophy, progressive facial.1051
Hemicrania 1021
Hemiplegia 974
Hemoglobinuria 815
Hemorrhagic diseases of the new-born 335
Hemopericardium 487
Hemophilia 336
Hemoptysis 426, 427
and hematemesis, differential points 663
diagnosis of 427
etiology of 426
parasitic 1086
pathology of 426
prognosis of 427
symptoms of 428
treatment of 428
Hemorrhage, cerebral 974
into spinal cord 912
pulmonary 429
spinal meningeal 910
vesical 880
Hemorrhagic infarct of the bowels 703
Hemorrhoids 724
treatment of 725
Henoch's purpura 333
Hereditary ataxia 945
Heredity in gout 307
in tuberculosis 196, 198
Hob-nail liver 777
Hodgkin's disease 360
Huntingdon's chorea 1032
Hydatid cyst of lung 446
Hydrocephalus, chronic 968
Hydrogen peroxide, in asthma... 417
Hydronephrosis 869
Hydropericardium 486
Hydrophobia 262
diagnosis of 264
etiology of 263
pathology of 263
prognosis of 265
symptoms of 263
treatment of 360
inoculative treatment of 265
prophylactic 265
Hydrotherapy in pneumonia 191
in typhoid fever 29
Hydrothorax 467
Hyperacidity 672
Hyperacusis 900
Hyperchlorhydria 672
diagnosis of 674
symptoms of 673
treatment of 674-677
Hyperemia, active of kidneys.... 808
cerebral 969
of lungs, active 422
mechanical 423
passive 423
spinal 915
of spleen 791
passive, of kidneys 811
Hyperesthesia of stomach 680
Hyperorexia 680
Hyperosmia 894
Hypertrophy, localized 1056
of heart 533
osteo-arthropathy 1050
of liver 778
pseudo-muscular 956
Hyperostosis cranii 1051
Hypoglossal nerve, diseases of.. 907
Hypostatic congestion of lungs in typhoid 18
Hysteria 1004
accidents of 1007
cataleptic stage of 1009
grand attack in 1007
hemiplegia in 1010
major 1013
motor stigmata in 1006
mental stigmata in 1006
symptoms of 1005
treatment of 1014
Hysteric contractures 1010
Ichthyismus 1072
Icterus 742
neonatorum 743
Incontinence of urine 831
Indicanuria 817
Infectious diseases 1
Influenza 171
bacteriology of 171
diagnosis of 173
etiology of 171
complications in 174
pneumonia in 175

    manner of invasion 171
    modes of conveyance 171
    predisposing causes of 171
    prognosis of 174
    sequelae of 178
    symptoms of 172
    treatment of 173
    types in 172
Insufficiency of aorta 505
    mitral 498
    tricuspid 512
    pulmonary 517
Intermittent fever (see *malarial fever*) 54
Interstitial pneumonia 430
Intestinal obstruction 706
    internal strangulation 706
    fecal obstruction 709
    intussusception 707
    stricture and morbid growth 708
    obstruction from abnormal contents 709
    chronic 711
    ulceration 704
    volvulus 708
    treatment of 714
Intestine, carcinoma of 722
    constipation 715
    paralysis of 709
    treatment of 717
    disturbances of motility 729
    enteralgia 728
    appendicitis 732
    secretory disturbances of ... 728
    sensory disturbances of 729
    neuroses of 728
    nervous diarrhea 729
Intestines, diseases of 681
    diagnostic technique 681
Intussusception 707
Irodoplegia 896
Jacksonian epilepsy 999
Jaundice 742
    catarrhal 743
    hematogenous 742
    hepatogenous 742
    malignant 742
Jiggers (see *Chiggers*) 1082
Jumpers 1036
Justus' blood-test for syphilis... 251
Kernig's sign 97
Kidneys, amyloid 826
    carcinoma of 873
    cystic 872
    floating 806
    hyperemia of, active 808
        passive 810
    in diabetes 285
    in diphtheria 131
    in gout 310
    in typhoid fever 9
    large white 848
    mottled 848
    mobility of 806
    movable 807
    rhabdomyoma of 873
    sarcoma of 873
    small white (see *secondary contracted*) 848
    syphilis of 250
    tuberculosis of 230
Klebs-Loeffler bacillus 128
Klemperer's test for motor stomach function 633
Koplik's sign, of measles 159-160
Lactic acid 630
La Grippe (see *influenza*) 171
Landry's paralysis 933
Laryngeal diphtheria 133
Laryngismus stridulus 393
Laryngitis, acute 388
    chronic 391
    edematous 399
    membranous 133
    spasmodic 393
    syphilitic 397
    tuberculous 395
Larynx, diseases of 388
    new growths in 399
    paralysis of 903
    spasm of 903
Lathyrismus 1073
Lead poisoning 1067
Leprosy 254
    anesthetic form of 255
    bacteriology of 254
    diagnosis of 256
    etiology of 254
    pathology of 255
    prognosis of 256
    symptoms of 255
    treatment of 256
    tubercular form of 254
Leptomeningitis, acute spinal ... 919
    chronic spinal 920
    brain 963
Leube's test for stomach motor function 632
Leukemia 355
    blood in 358
    diagnosis 359
    etiology of 355
    lymphatic 357
    pathology of 355
    prognosis of 359
    spleen in 356
    spleno-medullary 356
    symptoms of, general 357
    treatment of 359
Leukocythemia 355
Lipomatosis, symmetrical 1056
Lithemia 315
    diagnosis and prognosis 316
    symptoms of 316

  - treatment of 317
- Lithuria 819
- Little's disease 984
- Liver and bile tract, diseases of. 741
  - abscess of liver 766
  - abnormalities in shape and position 741
  - amyloid liver 762
  - acute yellow atrophy of...... 769
  - catarrhal jaundice 743
  - cancer of liver 773
  - calculous cholecystitis 749
  - carcinoma of gall-bladder, etc. 753
  - cholecystitis, acute infectious 747
  - cirrhosis of liver 776
  - fatty liver 759
  - icterus 742
  - hypertrophic cirrhosis ..778, 779
  - jaundice 742
  - in phosphorus poisoning..... 771
  - perihepatitis 764
  - stenosis of the bile ducts.... 754
  - vascular affections of the liver 755
    - passive hyperemia 756
    - pylethrombosis 757
    - pylephlebitis, suppurative. 758
  - syphilis of 247
  - tuberculosis of 229
- Lobar pneumonia 178
- Lockjaw (see *tetanus*) 266
- Locomotor ataxia 934
- Ludwig's angina 599
- Lumbago 271
- Lumbar puncture in meningitis.. 98
- Lumpy jaw (see *actinomycosis*). 258
- Lungs, diseases of 422
  - abscess of 441
  - broncho-pneumonia 432
  - carcinoma of 444
  - circulatory disturbances of.. 422
  - chronic interstitial pneumonia 431
  - cirrhosis of 430
  - compression of 450
  - edema, pulmonary 424
  - embolism, pulmonary 429
  - emphysema 435
  - fever of 178
  - flukes in 1086
  - gangrene of 440
  - hydatid cyst of 446
  - hyperemia of, active 422
    - hypostatic 423
    - mechanical 423
    - passive 423
  - pneumorrhagia 429
  - pneumonokoniosis 443
  - syphilis of 249
- Lymphadenoma (see *pseudo-leukemia*) 360
- Lymphatic glands in diphtheria.. 131
- Lymph-glands, tuberculosis of... 202
- Lymphoma, multiple malignant (see *pseudo-leukemia*) ... 360
- Lymphosarcoma, malignant (see *pseudo-leukemia*) 360
- Maidismus 1073
- Malarial fever 50
  - ague, cake in 54
  - algid form 56
  - bacteriology of 51
  - blood changes in 51
  - cachexia 58
  - comatose form 56
  - complications of 59
  - diagnosis of 60
  - etiology of 57
  - estivo-autumnal parasite of. 56
  - fever in 57
  - hematuria and hemoglobinuria 59
  - in typhoid fever 4
  - intermittent 54
  - masked intermittent 58
  - mosquitoes in 51
  - pathology of 54-57
  - pernicious malarial intermittent 55
  - plasmodium in 51
  - predisposing causes of 53
  - prognosis of 60
  - prophylaxis 61
  - quartan parasite in 52
  - remittent 57
  - tertian parasite in 57
  - treatment of 60
- Malignant diphtheria 132
- Malignant pustule (see *anthrax*) 260
- Malta fever 49
- Mammary glands, tuberculosis of 231
- Mania-a-potu 1062
- Measles 158
  - bacteriology of 158
  - complications in 160
  - diagnosis of 160
  - eruptive stage 159
  - etiology of 158
  - French (see *rubella*) 162
  - German (see *rubella*)...... 162
  - Koplik's sign 159
  - pathology of 158
  - prognosis of 161
  - symptoms of 158
  - treatment of 162
- Mediastinum, diseases of.....469-471
  - hemorrhage of 472
  - inflammation of 471
  - tumors of 470
- Méniére's disease 901

Meningitis, spinal 916
  cerebro-spinal 93
  tuberculous 965
Mercurialism 1070
Mesenteric glands in typhoid fever 17
Micrococcus lanceolatus 178
  melitensis 49
Micturition, neuroses of 880
Migraine 1021
Miliary fever 279
  tuberculosis 204, 214
Milk-sickness 278
Mind-blindness 895-983
Mitral incompetency 498
Mitral insufficiency 498
  diagnosis of 501
  physical signs of 501
  symptoms of 499
  regurgitation 498
    with aortic regurgitation.. 520
  stenosis 502
    diagnosis of 505
    pathology of 503
    physical signs of 504
    symptoms of 503
Morbus, maculosus neonatorum 336
Morphinism 1065
Morton's neuralgia 893
Mosquitoes in malarial fever.... 51
Motor oculi 896
  paralysis of 896
  spasms of 896
Mountain fever 40
Mucous colic 728
Multiple neuritis 884
Multiple cerebro-spinal sclerosis. 948
Mumps 169
  complications in 170
  diagnosis of 170
  etiology of 169
  fever in 170
  symptoms of 170
  treatment of 170
Muscular atrophy, progressive... 951
Muscular rheumatism 270
Mushroom poisoning 1074
Myalgia 270
Myiasis 1090
Myoclonia 1033
Myelitis, acute transverse 921
  chronic 926
Myocarditis 544
  acute 544
  chronic 546
    physical signs of 548
    pathology of 546
    symptoms of 547
    treatment of 549
Myosis 896
Myositis acute multiple 273
  progressive ossifying 274
Myotonia 1043
Myxedema 376
  diagnosis of 379
  etiology of 376
  operative 379
  pathology of 376
  prognosis of 379
  symptoms of 377
  treatment of 379
  with cretinism 378
Nematodes 1075
Nephritis, acute 834
  diagnosis of 837
  etiology of 834
  pathology of 835
  prognosis of 838
  symptoms of 836
  treatment of 838
  chronic 846
    non-indurative (parenchymatous) 847
    pathology of 848
    prognosis of 851
    treatment of 851-859
    uremia in 849
    symptoms of 849
    urine in 849
    chronic indurative (interstitial) 858
    circulatory disturbances in 861
    diagnosis in 863
    digestive symptoms in 862
    etiology of 859
    nervous symptoms in 862
    pathology of 859
    prognosis of 863
    retinitis in 862
    respiratory system in..... 862
    symptoms of 860
    treatment of 864-867
    urinary symptoms in 861
Nephrolithiasis 828
Nephydrosis (see *hydronephrosis*) 869
Nerve, brachial plexus 908
  cervical plexus 908
  eighth (auditory) 900
  fifth (trigeminus) 897
  fourth (patheticus) 896
  lumbar plexus 909
  motor oculi 896
  ninth (glosso-pharyngeal)... 903
  optic 895
  olfactory 894
  phrenic 908
  sacral plexus 909
  seventh (facial) 898
  sixth (abducens) 896
  spinal accessory 905
  tenth (pneumogastric) 903
  third (motor oculi) 896
  twelfth (hypoglossal) 907

- Nervous complications in typhoid 18
  - diarrhea 729
  - dyspepsia 669
- Neuralgia 888
  - cervico-brachial 892
  - cervico-occipital 891
  - crural 892
  - femoral 892
  - intercostal 891
  - Morton's neuralgia 893
  - obturator 892
  - of extremities 892
  - of neck and trunk 891
  - plantar 893
  - trifacial 889
  - visceral 893
- Neurasthenia 1016
  - symptoms of 1017
  - treatment of 1019
- Neuritis 883
  - focal 883
  - multiple 884
  - of optic nerve 895
  - peripheral in diabetes 288
  - pernicious multiple neuritis 886
- Neuromas 887
- Neuroses, fatigue 1040
  - cardiac 557
  - of the bladder 880
  - of the intestine 728
  - of the stomach 665
- New-born, hemoglobinuria of 335
  - hemorrhagic diseases of 335
  - morbus maculosus of 336
  - syphilis hemorrhagica of 335
- Night-blindness 895
- Night-sweats, in tuberculosis 215
  - treatment of 240
- Ninth nerve 903
- Noma 549
- Nose, diseases of 381
- Nose-bleed (see *epistaxis*) 387
- Nytalopia 895
- Obesity 338
  - treatment of 340
- Occupation neuroses 1040
- Oppler-Boas bacillus 633
- Optic atrophy 895
  - nerve, choked disk of 895
  - neuritis of 895
  - perineuritis of 895
  - tract, lesions of 895
- Osteo-arthropathy 1050
- Ovaries, tuberculosis of 231
- Oxaluria 821
- Oxyuris vermicularis 1076
- Pachymeningitis, spinal externa 916
    - interna 917
  - cerebral externa 962
  - cerebral interna 963
- Palpitation of heart 557
- Palsy, Bell's 899
  - cerebral, of children 983
  - shaking 1036
- Pancreas, diseases of 783
  - calculi 790
  - carcinoma 786
  - cysts of 788
  - hemorrhage into 783
  - in diabetes 282
- Pancreatitis, acute 783
  - chronic 785
- Paranephric abscess 871
- Paralysis, acute ascending 933
  - agitans 1036
  - Bell's 899
  - bulbar 954
  - family periodic 1042
  - general, of insane 990
  - glosso-labio-laryngeal 954
  - intra-ocular 896
- Parkinson's disease 1036
- Osteomalacia 324
  - diagnosis of 325
  - pathology of 324
  - prognosis of 325
  - treatment of 325
- Paralysis, hysteric 110
  - muscular pseudo-hypertrophic 95
  - of spinal accessory nerve 905
  - spinal, Brown-Séquard's 959
- Paramyoclonus multiplex 1033
- Paraplegia, ataxic 942
  - hereditary spastic 947
  - senile, acute and chronic 932
- Paratyphoid fever 39
- Paresis, general 990
- Parkinson's disease 1036
- Parotiditis (see *parotitis*) 169
- Parosmia 394
- Parotitis 169
  - metastatic 601
  - Parrot's ulceration 574
- Patheticus 896
- Pellagra 1073
- Pepsin, test for 631
- Pericarditis 473
  - acute plastic 474
  - adhesive 484
  - chronic 484
  - fibrinous 474
  - purulent 483
  - sero-fibrinous 476
- Perihepatitis 764
- Paranephric abscess 871
- Perineuritis, optic 895
- Peristalsis, diminished, of stomach 679
- Peristaltic unrest 679
- Peritoneum, carcinoma of 801
  - dropsy of 801
  - tuberculosis of 228
  - new growths in 801

- Peritonitis, acute 793
  - adhesive 798
  - circumscribed 795
  - chronic 798
    - localized 798
    - tuberculous 799
  - in typhoid fever 10, 17, 35
  - proliferative 799
- Peritonsillar abscess 603
- Pertussis, (see *whooping-cough*) 164
- Pestis, major 91
  - minor 91
- Petit mal 998
- Peyer's patches in typhoid..... 6
- Pharyngeal diphtheria 132
- Pharyngitis 607
  - acute catarrhal 607
  - chronic. 608
  - gangrenous 610
  - phlegmonous 610
  - treatment of 612
- Pharynx, ulceration of........ 609
  - tuberculosis of 609
  - syphilis of 609
- Phosphaturia 822
- Phosphorus-poisoning, liver in.. 771
- Phrenic nerve 908
- Phthisis, acute pneumonic...... 210
- Plague 91
  - bacillus of 91
  - bubo in 92
  - diagnosis of 93
  - etiology of 91
  - modes of transmission...... 91
  - protective inoculation in.... 93
  - prognosis of 93
  - pneumonic form 92
  - septicemic form 92
  - symptoms of 92
  - treatment of 93
  - varieties of 91
- Plasmodium malariae 51
- Pleurae, diseases of 446
- Pleurisy 446
  - acute plastic 447
    - diagnosis of 448
    - etiology of 447
    - pathology of 448
    - prognosis of 448
    - treatment of 449
  - chronic dry 463
    - with effusion 462
  - diaphragmatic 453
  - encysted 454
  - hemorrhagic 453
  - interlobar 454
  - neoplasms in 468
  - sero-fibrinous 449
    - diagnosis of 455
    - etiology of 449
    - pathology of 450
    - physical signs of 451
    - special forms of 453
    - symptoms of 450
    - treatment of 457-459
  - tuberculous ....
    ....449, 215, 227, 447, 453
- Pleuritis 446
- Pleurodynia 271
- Pleuro-typhoid 21
- Plumbism 1067
- Pneumogastric nerve 903
- Pneumonia, lobar 178
  - age in 179
  - alcohol in 180, 188, 191
  - bacteriology of 179
  - bronchitis in 185
  - broncho-pneumonia in 432
  - digestive disturbances in... 184
  - duration of 181
  - cardiac features in 188
  - cerebral symptoms in 184
  - chronic interstitial 430
  - clinical varieties in......... 186
  - complications in 185, 186
  - coughs in 183
  - diagnosis of 187
  - edema of lungs in 185
  - endocarditis in 186
  - etiology of 179
  - fever in 183
  - lesions of other organs in.. 181
  - leukocytosis in 183
  - meningitis in 186
  - nervous symptoms in ...184, 188
  - pain in 183
  - pathology of 186
  - pericarditis in 186
  - phthisis 187
  - physical signs in 184
  - pleurisy in 188
  - predisposing causes in..179, 185
  - prognosis of 188
  - pulse in 182
  - respiration in 182
  - typhoid in 187, 196
  - treatment of 188
    - general management 189
    - local applications 190
    - stimulants 191
    - hydrotherapy 191
    - special features 192
    - medicines in 193
- Pneumonitis (*see lobar pneumonia*) 178
- Pneumonokoniosis 443
- Pneumopericardium 487
- Pneumorrhagia 429
- Pneumothorax 464
  - Biemiers' test 466
  - coin-test in 466
  - diagnosis of 466
  - physical signs in 465
  - symptoms of 465

treatment of 467
Wintrich's sign 466
Podagra (*see gout*)........... 306
Poisoning by fish..............1072
by grain 1073
by infected milk 1071
by meat 1072
by mushrooms 1074
Polioencephalitis, superior acute. 972
inferior acute 972
Poliomyelitis anterior 929
Poliomyositis, acute 273
Probable infectious diseases..... 270
muscular rheumatism 270
acute multiple myositis 273
chronic articular rheumatism 274
foot and mouth disease..... 280
glandular fever 281
Weil's disease 276
febricula 277
milk-sickness 276
miliary disease 279
Progressive muscular atrophy... 951
Propeptone, test for 631
Pseudo-angina 570
Pseudo-diphtheria 128
Pseudophobia 265
Pseudo-leukemia 360
diagnosis of 362
etiology of 360
pathology of 360
prognosis of 363
symptoms of 361
treatment of 363
Psoriasis, buccal 599
lingual 598
Psorospermiasis 1088
Pseudo-muscular hypertrophy... 956
Ptomaine poisoning 1071
Ptyalism 599
Pulmonary apoplexy 429
atelectasis 432
edema 424
embolism 429
incompetency 517
regurgitation 517
stenosis 517
Pulse in typhoid fever.......... 11
Pulse-respiration, ratio in pneumonia 182
Pulsus bigeminus 565
paradoxus 565
trigeminus 565
Purpura 331
arthritic 332
diagnosis of 334
hemorrhagica 333
Henoch's 333
primary 332
prognosis of 334
secondary 331, 332
simple 332
treatment of 334
Pustule, malignant (*see anthrax*) 260
Pyelitis 866
Pyelonephritis 866
Pyemia 112
bacteriology of 112
diagnosis of 114
etiology of 112
pathology of 113
prognosis of 114
symptoms of 113
treatment of 114
Pylephlebitis, suppurative 754
Pylethrombosis 757
Pyloric relaxation 679
Pylorospasm 679
Pyonephrosis 866
Pyopneumothorax 464
Pyrosis 678
Pyuria 817
Quartan fever, ameba of 52
Quincke's lumbar puncture...... 98
Quinsy 603
Rabies (*see hydrophobia*)...... 262
Race in tuberculosis 199
Rachitic paralysis 319
rosary 320
Rachitis 318
diagnosis of 321
etiology of 318
pathology of 319
prognosis of 321
symptoms of 320
treatment of 321
Ragpicker's disease 262
Ray fungus 258
Raynaud's disease 1053
Rectum, syphilis of 249
Regurgitation, aortic 505
mitral 498
pulmonary 517
tricuspid 512
Relapsing fever 46
complications of 48
diagnosis of 48
prognosis of 48
treatment of 49
Renal calculi 828
colic, treatment of 833
cyst 872
Rennet ferment 631
zymogen 632
Respiratory system, diseases of.. 381
Retention of urine 882
Retina, hemorrhage into 894
Retinitis 894
albuminuric 894
syphilitic 894
pigmentary 894
Retropharyngeal abscess 611

- Rhabdomyoma, of kidney 873
- Rheumatic fever 115
  - bacteriology of 117
  - cardiac involvement in 118
  - chorea 117
  - complications in 119
  - diagnosis of 120
  - etiology of 116
  - endocarditis in 117
  - joint involvement in 118
  - myocarditis in 120
  - nervous symptoms in 119
  - nodules in 120
  - pathology of 117
  - pericarditis in 119
  - prognosis in 121
  - skin in 119
  - symptoms of 117, 119
  - treatment of 121
    - local 122
    - internal 122
- Rheumatism, chronic articular 274
- Rheumatoid arthritis (see *arthritis deformans*) 300
- Rhinitis, acute 381
  - chronic 383
- Rice-water stools of cholera 77
- Rickets (see *rachitis*) 318
- Rock fever (see *Malta fever*) 49
- Rotheln (see *rubella*) 162
- Round-worm 1075
- Rubella 162
  - complications in 164
  - diagnosis of 164
  - etiology of 163
  - prognosis of 164
  - symptoms of 163
  - treatment of 164
- Rubeola notha (see *rubella*) 162
- Rumination 678
- Salivation 599
- Sapremia 110
- Sarcoma of the kidney 873
  - of liver 772
- Saturnism 1067
- Sausage poisoning 1072
- Scarlatina (see *scarlet fever*) 149
- Scarlet fever 149
  - bacteriology of 149
  - cardiac complications in 153
  - complications in 152, 153
  - desquamation in 151
  - diagnosis of 153
  - eruption in 150
  - etiology of 149
  - leukocytosis in 151
  - malignant 152
  - modes of conveyance 149, 150
  - nephritis in 157
  - nervous symptoms of 151, 153
  - paralysis in 153
  - pathology of 150
  - predisposing causes in 150
  - prognosis of 154
  - symptoms in 150
  - treatment of 154
    - disinfection 155
    - general 154
    - local 155
    - prophylactic 154
    - remedies in 155-157
  - rash in 149-151
  - tongue in 151
  - types of 151, 152
  - urine in 151
- Schoenlein's disease 332
- Schott baths, in heart disease 526
- Sciatica 892
- Scleroderma 1055
- Sclerosis, amyotrophic lateral 953
  - arterial 574
  - combined 944
  - multiple, cerebro-spinal 948
  - of liver 777
  - posterior 934
  - primary lateral 943
- Scorbutus 326
  - bacteriology of 326
  - diagnosis of 328
  - infantile 329
  - pathology of 326
  - prognosis of 328, 331
  - symptoms of 327, 331
  - treatment of 329, 331
- Scrofula (*see tuberculosis of lymph-glands*) 202
- Scurvy (*see scorbutus*) 326
- Septicemia 109
  - antistreptococcus serum in 112
  - bacteriology of 109
  - diagnosis of 111
  - etiology of 109
  - pathology of 110
  - prognosis of 111
  - symptoms of 110
  - treatment of 111
- Sero-fibrinous pleurisy 449
- Sero-reaction in typhoid 23
- Seventh nerve 898
- Ship-fever (*see typhus fever*) 40
- Siderosis 443
- Sixth nerve 896
- Skin in typhoid fever 14
- Skoda's resonance in sero-fibrinous pleurisy 452
  - in pneumonia 184
- Sleeping sickness 1088
- Smallpox (see *variola*) 138
  - black 141
- Smell, sense of 894
- Spasm, habit 1035
  - of larynx 903
  - of stomach 679
  - saltatoric 1036

Spasmodic laryngitis............ 393
Spastic progressive muscular atrophy 953
Spinal accessory nerve, paralysis of 906
  cord, anemia of............ 916
    compression of 921
    hemorrhage into 910
    hyperemia of 915
    tumors of 958
Spirillum, Obermeieri 46
Spleen, amyloid degeneration of. 791
  dislocation of 799
  hemorrhagic infarct of...... 792
  hyperemia of 791
  in leukemia 356
  in lobar pneumonia 181
  in typhoid fever 8, 17
  new growths of........... 791
Splenic fever 260
Splenitis 792
Sprue 703
Sputum in lobar pneumonia..... 183
  in tuberculosis 215
St. Anthony's dance............1024
  fire 103
St. Vitus' dance 1024
Staticus lymphaticus 364
  etiology of 364
  pathology of 364
  symptoms of 365
  treatment of 365
Status epilepticus 998
Stenocardia 567
Stenosis, aortic 509
  bronchial 413
  mitral 502
    with mitral insufficiency... 518
    with aortic stenosis 518
    with aortic regurgitation.. 519
  of bile ducts 754
  pulmonary 517
  tricuspid 515
Stomach, carcimona of.......... 659
  acute catarrhal gastritis.... 639
  chronic catarrhal gastritis.. 644
  diagnostic technique in..... 623
  dilatation of 635
  diminished peristalsis of... 679
  displacement of 633
  examination of gastric contents 627, 633
    for absorptive power..... 632
    for acetic acid 631
    for diminished acidity ... 629
    for fatty or volatile acids. 631
    for free HCl 629, 630
    for lactic acid 630
    for motor function 632
    physical 623
    for propeptone 631
    for peptone 631
    for erythrodextrin, etc. .. 632
    for total acidity 629
    tropeolin O. O. 630
  hyperesthesia of 680
  hyperorexia 680
  in cholera 76, 77
  in typhoid fever 14
  increased peristalsis of 678
  malposition of 633
  neuroses of 655
    of motility 678
    of secretion 679
    of sensation 680
  spasm of 679
  tuberculosis of 225
  ulceration of 653
Stomatitis 590
  aphthous 591
  catarrhal 590
  epidemic (see *foot and mouth disease*) 280
  fetid 591, 593
  follicular 591
  gangrenous 594
  mercurial 594
  mycotic 591
  parasitic 591
  syphilitic 594
  treatment of 596
  ulcerative 593
Streptococcus erysipelatis, pneumoniae 103
Strongyloides intestinalis 1083
Stricture of the intestine........ 708
Strychnine poisoning and tetanus, differentiated 268
Subnormal gastric secretion.... 677
Sunstroke 1057
Suprarenal capsule, diseases of. 368
  glands, role of in diabetes.. 283
Swamp fever (see *malarial fever*) 50
Sweating sickness (see *malarial fever*) 50
Sydenham's chorea 1024
Syphilis 240
  bacteriology of 241
  contagion of 240, 241
  congenital symptoms of..... 244
    early post-natal symptoms. 244
    in new-born 244
  etiology of, general......... 241
  diagnosis of, general....... 250
  Justus' blood test for....... 251
  modes of infection of....... 241
  of alimentary tract.......... 248
  of brain and cord.......245, 246
    etiology of 246
    symptoms of 246
  of circulatory system 250
  of joints 250
  of kidneys 250

of liver 247
  diagnosis of 248
  symptoms of 247
and cancer, differential points 248
of lungs 249
  symptoms of 249
of spleen 248
of testicles 250
pathology of, general 241
symptoms of 242
  acquired form 242
  congenital form 244
treatment of 251
  by fumigation 253
  by inunctions 252
  hypodermatic 253
  prophylactic 251
  remedies in 252, 254
visceral 245
  etiology of 246
  symptoms of 246
Syringomyelia 961
Tabes dorsalis 934
  mesenterica 203
Tachycardia 560, 904
  in exophthalmic goiter 1044
Tapeworm 1083
Tenia, lata 1084
  confusa 1083
  echinococcus 1084
  mediocanellata 1084
  nana 1083
  solium 1084
Tarsalgia 893
Tenth nerve 903
Tertian fever, intermittent, ameba of 57
Test-breakfast, of Ewald and Boas 627
Testes, tuberculosis of 231
Testicles, syphilis of 250
Tetanus 266
  acute 267
  and strychnin poisoning 268
  bacteriology of 266
  cephalic 268
  chronic 268
  diagnosis of 268
  etiology of 266
  fever in 268
  immunity to 267
  modes of infection 267
  pathology of 267
  prognosis of 269
  symptoms of 267
  treatment of 269
Tetanus antitoxin 269
Tetany 1038
Thermic fever 1057
Third nerve 896
Thomsen's disease 1043
Thrombosis in typhoid fever 19
  of brain 979
  of portal vein 757
Thrush 591
Thymus gland, diseases of 472
Thyroid gland, diseases of 372
Thyroiditis 372
Tic douloureux (see *trifacial neuralgia*) 889
  general 1035
  tinnitus aurium 900
Tongue in diabetes 287
  tuberculosis of 225
Tonsil, tuberculosis of 225
Tonsillitis, acute 602
  catarrhal 602
  parenchymatous 603
  chronic 605
  follicular 603
    and diphtheria, differential points of 135
  necrotic 604
Tonsils, diseases of 602
Töpfer's test for HCl in gastric contents 630
Torticollis 271, 905
Traube's semilunar space 625
Trematodes 1085
Trembles 278
Trichniasis 1076
Trichocephalus dispar 1083
Tricuspid incompetency 512
  insufficiency 512
  regurgitation 512
  stenosis 512
Trigeminus 897
Trismus (see *tetanus*) 266
Trousseau's sign 1039
Trypanosomiasis 1087
Tuberculin test for tuberculosis 233
Tuberculosis 196
  cavity in 221
  changes in the tubercle 201
  contagion in 198
  diagnosis of 232
  etiology of 196
  environment in 199
  hemoptysis in 200, 217
  heredity in 196, 198
  in animals 197
  inhalation infection 198
  Koch's tuberculin test 233
  local conditions in 199
  miliary tubercles in 204, 214
  modes of conveyance 198
  night sweats in 215, 240
  number of bacilli 197
  occupations in 199
  pathology, general 200
  predisposing causes of 198, 199
  previous infections 199
  prognosis of 233

Tetan's test 233
traumatism in 200
treatment of 234
  antiseptic inhalations 236
  climatic 237
  internal parasiticides 236
  dietetic 238
  special remedies 238
  homeopathic remedies 239
varieties of 202
acute tuberculosis 204
  diagnosis of 209
  prognosis of 209
  symptoms of 208
meningeal form of 207
  pathology of 207
pulmonary form 206
  diagnosis of 206
  symptoms of 206
typhoid form 205
  diagnosis of 205
  symptoms of 205
acute pneumonic 210
bronchopneumonic 211
symptoms of 210
fibroid phthisis 223
  course and duration of 224
  concurrent infections 224
  symptoms of 224
chronic pulmonary 212
  diagnosis of 222
  pathology of 212
  physical signs of 219
  symptoms of 215
  of the alimentary canal 225
  symptoms of 226
  of the lymph-glands 202
  diagnosis of 202
  symptoms of 202
  of various tissues 226
of pericardium 227
of peritoneum 228
of liver 229
of mesenteric glands 203
of genito-urinary system 231
of mammary glands 231
of brain 231
of serous membranes 226
of heart and blood vessels 232
Tuberculous adenitis 204
  meningitis 707, 965
  diagnosis of 967
  pathology of 965
  prognosis of 967
  symptoms of 966
  treatment of 967
  pleuritis 227
Tumor of brain 985
  of larynx 399
  of mediastanum 471
  of peritoneum 801
  of pleurae 468
  of spinal cord 958
Tussis convulsiva (see *whooping cough*) 164
Twelfth nerve 907
Typhoid fever 1
  abscess in 9
  albuminuria in 20
  ambulatory form 12, 21
  antisepsis in 30
  appendicitis in 15, 25
  arteritis in 10
  atypical forms of 21
  bacillus of 2
    methods of entrance 6
    in blood 2
    in urine 2, 20
  bathing in 20, 29
  bed sores in 14, 75
  blood changes in 10, 18
  blood vessels in 10
  boils in 14
  bone lesions in 20
  Brand bath in 29
    contra-indications to 30
  cardiac failure in 28
  in children 22
  circulatory disturbances in 18
  chills in 11
  cholecystitis in 17
  cholelithiasis in 17
  coma in 19
  complications of 21
  constipation in 15
  convulsions in 13
  cystitis in 20
  definition of 1
  delirium in 19, 33
  diagnosis of 23
  differential diagnosis of 24
  digestive disturbances in 14
  diarrhea in 15
  diazo reaction of urine in 20, 24
  diet in 27
  disinfection of discharges in 26
  endocarditis in 10, 18
  enemas in 32
  etiology of 1
  facial expression in 12
  fever in 12
  gangrene in 14
  genito-urinary complications in 20
  headache in 11, 18
  heart changes in 9, 18
  hemiplegia in 19
  hemorrhage in 16
    into brain 10
    treatment of 35, 39
  hepatic complications in 8, 17
  herpes in 14
  history of 1
  hydrotherapy in 29

hypostatic lung congestion in 18
incubation period of........ 10
inoculation, preventive...... 26
infarction of lung in....... 9
insomnia in 33
jaundice in 17
joint complications in...... 20
kidneys in 9
leukocytosis in 10, 18
liver and bile tract in.....8, 17
lungs in 9
malaria in 4
meningitis in 24
mental condition in......11, 19
mesenteric glands in....... 17
mode of conveyance in..... 4
mortality of 25
muscle changes in......... 20
myocarditis in 10, 18
nephritis in 20
nervous changes in......... 10
symptoms of 18
neuritis, peripheral, in...... 10
nose bleed in............... 11
occurrence, forms of....... 5
osteomyelitis in 20
parotitis in 15
pathology of 6
peliomata in 14
pericarditis in............10, 18
perforation in 16
  abdominal signs of....... 16
  treatment of 35
peritonitis in 10, 17, 35
pharyngitis 15
phlebitis in 10
pleurisy in 9
pneumonia in 9, 25, 35
prophylaxis in 26
prognosis of 25
pyemic conditions 20
pyuria in 20
recrudescence in 13, 22
relapses in 22
respiratory symptoms 17
remedies, general consideration of 36
renal diseases in........... 20
rosespots in 14
septicemia post-typhoid 20
skin eruption in............ 14
spleen, changes in........8, 17
stimulants in 28
stools in 15
subsultus tendinum in...... 19
sweating in 14
symptoms and course of.... 10
tache bleuâtre in........... 14
  cerebrale 14
temperature in 13
sero-reaction in 23
  thirst in 11
  thrombosis in 19
  tongue in 14, 19
  treatment of 26
    during convalescence 36
    special symptoms 32
  tympanites in 15, 34
  typhoid spine in............ 19
  ulcer in 7
  vomiting in 33
  Widal's test in............. 24
Typhus fever 40
  chill in 42
  complications of 44
  coma-vigil in 43
  definition of 40
  delirium in 43
  diagnosis of 44
  eruption in 43
  etiology of 41
  fever in 43
  heart in 44
  history of 41
  hypostatic lung congestion in 42, 44
  mortality in 45
  pathology of 42
  petechiae in 43
  prognosis of 45
  pulse in 42
  symptoms of 42
  treatment of 45
  siderans 44
  urine in 19
  varieties of 21
Typho-mania 19
Tyrotoxicon 1071
Uffelmann's test of gastric contents 630
Ulceration, neurotic 594
Ulcer of esophagus............ 621
Ulcers, duodenal 653
  gastric 653
  intestinal 714
Uncinariasis 1070
Undulant fever (see *Malta fever*) 49
Uremia 822
  in chronic nephritis........ 849
  symptoms of 823
  treatment of 825
Uremic amaurosis 824
Urine, pathologic states in...... 813
  acetonuria 819
  albuminuria 816
  albumosuria 817
  Beta-oxybutyricaciduria 819
  choluria 818
  chyluria 818
  cystinuria 822
  diaceticaciduria 819
  glycosuria 818

  hematuria 813
  hemoglobinuria 815
  indicanuria 817
  lithuria 819
  oxaluria 821
  phosphaturia 822
  retention of 882
  urobilinuria 818
Vaccination 146
  treatment of 147
Vaccinia 147
Valvular heart diseases, combined 518
  treatment of 521
  during stage of compensation 522
  during stage of non-compensation 524
Varicella 147
  and variola 143
  complications of 148
  diagnosis of 148
  etiology of 147
  prognosis of 148
  symptoms of 147
  treatment of 149
Variola 138
  and varicella 143
  abortive form 142
  bacteriology of 138
  complications 142
  diagnosis of 142
  eruption in 140
  etiology of 138
  fever in 139
  initial rash 140
  hemorrhagic form 141
  modified forms 141
  pathology of 139
  prognosis of 144
  symptoms of 139
  treatment of 144
    hydrotherapic 144
    of eruption 144, 145
  prophylactic 144
  remedies 145, 146
Varioloid 142
Vein, portal, thrombosis of 757
Venous thrombosis in typhoid fever 18
Vertigo 902
Vesiculae seminales, tuberculosis of 231
Visceral syphilis 245
Von Graefe's sign 1045
Volvulus 706
Vomiting, nervous 678
Weil's disease 276
Whooping cough 164
  bacteriology of 165
  complications in 166
  description of cough in 166, 167
  diagnosis of 166
  etiology of 165
  pathology of 165
  prognosis of 167
  sequelae of 166
  symptoms of 166
  treatment of 167
Widal reaction in typhoid 23
Williams' tracheal tone in serofibrinous pleurisy 452
Winckel's disease 335
Wintrich's sign of tuberculous cavities 220
Wool-sorter's disease (see *anthrax*) 260
Word-blindness 982
Word-deafness 982
Wound diphtheria 134
Writer's cramp 1041
Wry-neck, spasmodic 905
Xerostoma 600
Yellow atrophy of the liver 759
Yellow fever 81
  albuminuria in 84
  bacteriology of 81
  diagnosis of 84
  etiology of 81
  gastric hemorrhage 84
  jaundice in 83
  mosquito in 82
  pathology of 83
  prognosis of 85
  prophylaxis 86
  remissions in 83
  symptoms of 83
  treatment of 85
  varieties of 84
Zygotes 52